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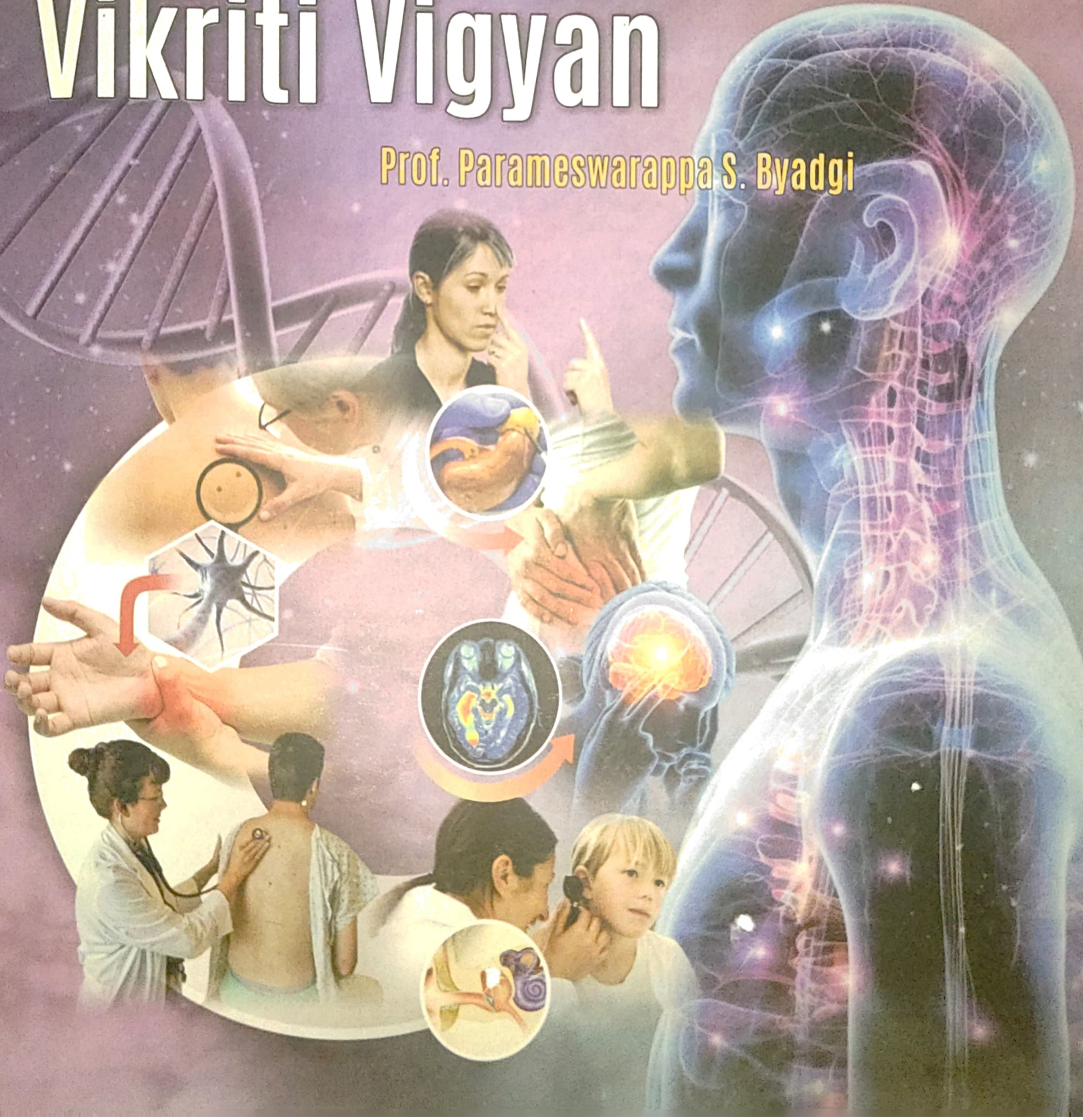
Parameswarappa's
Text book of
Roga Nidan evam Vikriti Vigyan

Prof. Parameswarappa S. Byadgi



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Parameswarappa's

Text Book of Roga Nidan & Vikriti Vigyan

National Commission for Indian System of Medicine (NCISM)

Vol. II

By

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CHAPTER 1

Agnimandya, Ajeerna, Anaha, Admana, Atopa

अग्निमान्द्य (Agnimāndhya)

Definition of Agnimāndhya

Dyspepsia (*Agnimāndhya*) is an indisposition in which food is not properly digested due to the impaired power of *Jaṭharāgni* (digestive juices).

प्रागभिहितोऽग्निरन्नस्य पाचकः । स चतुर्विधो भवति- दोषानभिपन्न एकः; विक्रियामापन्नस्त्रिविधो भवतिविषमो वातेन, तीक्ष्णः पित्तेन, मन्दः श्लेष्मणा, चतुर्थः समः सर्वसाम्यादिति ।

S.S. Su 35/24

Pācakāgni is stated to be responsible for digestion and metabolism. It is of four kinds based on the involvement of *doṣās*.

i) First variety is not associated with *doṣās* and it is called *samāgni* state and it is the physiological state of *Agni*. Remaining three varieties of *Agni* are the pathological ones and which are responsible for improper functions.
ii) *Viṣamāgni* state due to *vāta*.
iii) *Tikṣṇāgni* state due to *pitta*.
iv) *Mandāgni* state due to *kapha*.

समाग्नि (Samāgni)

Samāgni state of *Agni* is the condition in which *doṣās* are in equilibrium state and performs normal digestion and metabolism functions. C S Vi 1/25(4)

तत्र, यो यथाकालमुपयुक्तमन्नं सम्यक् पचति स समः समैर्दोषैः ।

S.S. Su 35/24

उद्गारशुद्धिरुत्साहो वेगोत्सर्गो यथोचित; ।
लघुता क्षुत्पिपासा च जीर्णाहारस्य लक्षणम् ।।

M.Ni 6/24 & C.S.Vi. 1/25(4)

It is characterized by :

1. यथाकालमुपयुक्तमन्नं सम्यक् पचति - Digestion of food within stipulated period of time, if person consume normal quantity of food
2. उद्गारशुद्धि - Belching which is free from morbidities
3. उत्साह - Enthusiastic to do all activities
4. वेगोत्सर्गो यथोचित - Proper elimination of natural urges without any difficulties
5. लघुता - Feeling of lightness all over the body
6. क्षुत्पिपासा - Timely hunger and thirst

विषमाग्नि (Viṣamāgni)

Viṣamāgni is the state in which improper digestion and metabolism takes place due to irregular function of *agni.*

यः कदाचित् सम्यक् पचति, कदाचिदाध्मानशूलोदावर्तातिसार- जठरगौरवान्त्रकूजनप्रवाहणानि कृत्वा स विषमः ।

S.S. Su 35/24

1. कदाचित् सम्यक् पचति - Some times *agni* performs normal functions
2. आध्मान - Flatulence
3. Metue - Abdominal pain
4. शूल - Upward movement of *vāta* inside the *koṣṭha*
5. अतिसार - Diarrhoea
6. जठरगौरव - Heaviness in abdomen
7. आन्त्रकूजन - Intestinal gurgling
8. प्रवाहणानि - Straining excercise for evacuation of stools.

तीक्ष्णाग्नि (Tīkṣṇāgni)

Tīkṣṇāgni is the state in which hyperfunctioning of digestion & metabolic activities observed.

यः प्रभूतमप्युपयुक्तमन्नमाशु पचति स तीक्ष्णः ।

S.S. Su 35/24

प्रभूतमप्युपयुक्तमन्नमाशु पचति - Quick digestion of food even after taking more quantity of food.

अत्याग्नि (Atyāgni)

स एवाभिवर्धमानोऽत्यग्निरित्याभाष्यते, स मुहुर्मुहुः प्रभूत- मप्युपयुक्तमन्नमाशुतरं पचति, पाकान्ते च गलताल्वोष्ठ- शोषदाहसंतापाञ्जनयति; । *S.S. Su 35/24*

1. एवाभिवर्धमानोऽत्यग्निरित्याभाष्यते - The *tikṣṇāgni* if increases greatly is called *atyāgni.*
2. स मुहुर्मुहुः प्रभूत-मप्युपयुक्तमन्नमाशुतरं पचति - Quicker digestion of food even after taking more quantity of food oftenly.
3. पाकान्ते च गलताल्वोष्ठशोषदाहसंतापाञ्जनयति - At the end of digestion it produces dryness, burning sensation and warmthness in the throat, palate and lips, .

नरे क्षीणकफे पित्तं कुपितं मारुतानुगम् ॥
स्वोष्मणा पावकस्थाने बलमग्नेः प्रयच्छति ।
तदा लब्धबलो देहे विरुक्षे सानिलोऽनलः ॥
परिभूय पचत्यन्नं तैक्ष्ण्यादाशु मुहुर्मुहुः ।
पक्त्वाऽन्नं स ततो धातूच्छोणितादीन् पचत्यपि ॥
ततो दौर्बल्यमातङ्कान्मृत्युं चोपनयेन्नरम् ।
भुक्तेऽन्ने लभते शान्तिं जीर्णमात्रे प्रताम्यति ॥
तृट्श्वासदाहमूर्च्छाद्या व्याधयोऽत्यग्निसंभवाः ।
तमत्यग्निं गुरुस्निग्धशीतैर्मधुरविज्जलैः ॥
C.S.Ci. 15/217-221

1. क्षीणकफे - Diminished state of *Kapha*
2. पित्तं कुपितं मारुतानुगम् स्वोष्मणा पावकस्थाने बलमग्नेः प्रयच्छति- *Pitta* along with *Vāta* strengthens the *Agni* in its own place i.e. *Grahaṇī* by its intrinsic heat.
3. तदा लब्धबलो देहे विरुक्षे सानिलोऽनलः । परिभूय पचत्यन्नं तैक्ष्ण्यादाशु मुहुर्मुहु - *Agni* become intensely powerful and immediately digest the frequently consumed food.
4. पक्त्वाऽन्नं स ततो धातूञ्छोणितादीन् पचत्यपि - After the digestion of food, it starts digesting the tissue elements like *Rakta* etc.
5. दौर्बल्य - Debility
6. आतङ्क - Suffers from many disorders
7. मृत्यु - Death
8. भुक्तेऽन्ने लभते शान्तिं जीर्णमात्रे प्रताम्यति - Feels comfort after taking food but soon become restless after it is digested.
9. The following conditions develop due to *Atyāgni.*

   तृट् - Thirst

   श्वास - Breathing

   दाह - Burning sensation

   मूर्च्छा - Fainting

Note- *Agni* is the heating attribute of *Pitta.*

Always there is close association exists between *Agni* and *Pitta* i.e. *Agni* is the substance which originates (*Janya*) and *Pitta* is the source of origin (*Janaka*). Due to this difference between the *Janaka* and the *Janya* the aggravated *Pitta* might even reduce the *Agni.* It is only is association with *Vāta, Pitta* becomes powerful and manifest *Atyāgni.*

मन्दाग्नि (Mandāgni)

Mandāgni is the state in which digestion process become weak and takes long time to digest the less quantity of food.

यस्त्वल्पमप्युपयुक्तमुदरशिरोगौरवकासश्वासप्रसेकच्छर्दिगात्रसदनानि कृत्वा महता कालेन पचति, स मन्दः । S.S. Su 35/24

1. यस्त्वल्पमप्युपयुक्त महता कालेन पचति - Slower digestion of food even after taking less quantity of food.
2. उदरगौरव - Heaviness in abdomen

   शिरोगौरव - Heaviness in head

   कास - Cough

   श्वास - Dyspnoea

   प्रसेक - Excessive salivation

   छर्दि - Vomiting

   गात्रसदन - Weaknes of the body

Alidated Questionnaire for assessment of *Agni* developed by Dr.P.S.Byadgi and Dr.Neera Saini

Validated Questionnaire for assessment of Samāgni

S.	Items	Yes	No.
1.	Do you eat food timely and whether it gets digested within stipulated time		
2.	Whether eructations are pure/ free from bad odour		
3.	Do you feel enthusiasm to perform daily activities		
4.	Do you pass urine and stool regularly		
5.	Do you feel lightness in body		
6.	Do you get hunger normally and timely		
7.	Do you get thirst normally and timely		

Validated Questionnaire for assessment of Viṣamāgni

S.	Items	Yes	No
1.	Have you any complaints of sometimes digestive power works properly sometimes may not		
2.	Do you feel flatulence		
3.	Have you complaints of pain in abdomen		
4.	Have you complaint of upward movement of *Vāta* in *Koṣṭha*		
5.	Are you suffering from diarrhoea		
6.	Have you complaint of heaviness in abdomen		
7.	Do you feel gurgling noise in abdomen		
8.	Do you feel like straining during defecation		

Validated Questionnaire for assessment of Mandāgni

S.	Items	Yes	No
1.	Do you feel that comparatively you eat less amount of food		
2.	Do you feel that your digestion is weak		
3.	Do you feel that small amount of food also digests after long time		
4.	Do you have complaint of heaviness in abdomen		
5.	Do you have complaint of heaviness in head		
6.	Do you feel excessive gurgling noise in abdomen		
7.	Have you complaints of flatulence		
8.	Are you suffering from cough		
9.	Do you feel breathlessness		
10.	Have you complaints of excess salivation		
11.	Are you suffering from vomiting or nausea		
12.	Do you feel dryness of mouth		
13.	Do you feel excessive weakness		
14.	Have you complaints of constipation		

Validated Questionnaire for assessment of Tiṣāgni

S.	Items	Yes	No
1.	Do you feel that large quantity of food digests very quickly		

Validated Questionnaire for assessment of Atyāgni/ Bhaṣmākagni

S.	Items	Yes	No
1.	Do you feel that large quantity of food digests very quickly, feel hungry again and again		
2.	Do you feel dryness of throat, palate and lips		
3.	Do you feel burning sensation in throat, palate and lips		
4.	Do you feel complaint of increase warmthness in throat, palate and lips		

The maximum number of points possible is 7. A classification of definitive *Samŕgni* requires a score of 4/7 or higher.

The maximum number of points possible is 8. A classification of definitive *Viṣamāgni* requires a score of 5/8 or higher.

The maximum number of point possible is 1. A classification of definitive *Tikṣṇāgni* requires a score of 1/1.

The maximum number of points possible is 14. A classification of definitive *Mandŕgni* requires a score of 8/14.

The maximum number of points possible is 4. A classification of definitive *Atyŕgni* requires a score of 3/4

Importance of Agni Vikṛti

विषमो वातजान् रोगांस्तीक्ष्णः पित्तनिमित्तजान्।
करोत्यग्निस्तथा मन्दो विकारान् कफसंभवान्॥

S.S. Su 35/25

- *Viṣamāgni* state gives rise to diseases due to *Vāta doṣa.*
- *Tīkṣṇāgni* state gives rise to diseases due to *Pitta doṣa.*
- *Mandāgni* state gives rise to diseases due to *Kaphadoṣa.*

उपशय/पथ्य (Upaśaya/pathya)

Useful diets in cases of Agnimāndhya

In cases of indigestion caused due to *Śleṣma*, one should first take the *Vamana* therapy. In cases of indigestion caused due to *Pitta*, one should first take the *Virecana* (purgation) therapy. In cases of indigestion caused due to *Vāta*, one should first take the *Svedana* (fomentation) therapy. In indigestion, different types of exercises and use of light meals are useful. Gruel of old rice of *Śāli* variety, soup of the *Mudga* pulse (kidney-beans), liquor, meats of deer, peacock, rabbit, *Lāvaka*, and the meat soups, small fishes, *Śāliñca* leaves, leaves of the shoots of bamboo branches, banana fruits, buttermilk, warm water, ginger, asafoetida, cooked leaves of *Vāstuka* (chenopodium) and *Cāṅgerī* (sorrel), radish, fruits of *Paṭola*, pomegranate, orange, *Āmalakī*, lemon, honey, butter, *Ghṛtam*, mustard oil, caraway seeds, black pepper, cooked leaves of fenugreek, ginger pieces, coriander seeds, curd, betel leaves, and *Kāravellaka* (bitter gourd) are also very useful. (B.R. 10/303-311)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Harmful measures for the patients of Agnimāndhya : Strong *Virecana* (purgation), taking cold water, Suppression of natural urges, waking up to late night-hours, blood-letting through leeches, taking *Śamī* variety of rice, eating before digestion of the previous food, taking irregular meals, drinking excess water, fish, blackberry, potato, milk and palm fruits are harmful for the patient of dyspepsia. One should also avoid the incompatible food items and the items that are heavy to digest and cause constipation. (B.R. 10/312-315).

अजीर्ण (Ajīrṇa)

Introduction

Diminished *Jaṭharāgni* (digestive juices) produced by some physical factors like excessive intake of food and water, irregularities in diet and sleep, and mental factors such as the envy, fear, anger etc. leads to a state in which the patient is unable to digest even *Sātmya* (wholesome) and *Laghu* (light) meals taken in time and this indisposition is identified as *Ajīrṇa.*

पर्याय (Paryāya)

Apāka and *Avipāka* are the synonyms of *Ajīrṇa.*

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

Dyspepsia is defined as having one or more symptoms of epigastric pain, burning, postprandial fullness, or early satiation.

Indigestion is pain or discomfort after eating, during the digestion of food.

अजीर्ण-न जीर्ण नञ् समासः। अपाकः तत्पर्य्यायः।

Śabdakalpdruma

Ajīrṇa is a negative compound word, which means indigestion. *Apāka* is the synonym of *Ajīrṇa.*

अजीर्णः-जठरानलमान्द्येन भुक्तन्नादरोके, रोगभेदः।

Vācaspatyam

Indigestion/incomplete digestion of ingested food due to *Jaṭhārānalamāndhya (Agnimāndhya)* is called *Ajīrṇa.*

न जीर्यति सुखेनान्नं विकारान् कुरुतेऽपि च।
तदजीर्णमिति प्राहुरतन्मूला विविधा रुजः॥

Si.Ni. 5/245

The indigestion of ingested food associated with various disorders & discomfort is known as *Ajīrṇa* and it is the root cause for *Vividhā Rujaḥ* (different kinds of diseases).

अनात्मवन्तः पशुवद्भुञ्जते येऽप्रमाणतः।
रोगानीकस्य ते मूलमजीर्णं प्राप्नुवन्ति हि॥

M.Ni. 6/14

The one who eats large quantity of food imprecisely like cattle will develop this disease *Ajīrṇa* and it is the root cause for many other diseases.

अजीर्ण हेतु (Ajīrṇa hetu)

अभोजनादजीर्णातिभोजनाद्विषमाशनात् ।
असात्म्यगुरुशीतातिरुक्षसंदुष्टभोजनात् ॥
विरेकवमनस्नेहविभ्रमाद्व्याधिकर्षणात् ।
देशकालर्तुवैषम्याद्वेगानां च विधारणात् ॥
दुष्यत्यग्निः । *C.S.Ci. 15/42-44*

अत्यम्बुपानाद्विषमाशनाद्वा सन्धारणात् स्वप्नविपर्ययाच्च ।
S.S.Su. 46/507

अनात्मवन्तः पशुवद्भुञ्जते येऽप्रमाणतः ।
रोगनीकस्य ते मूलमजीर्णं प्राप्नुवन्ति हि ॥
M.Ni. 6/14

Causative factors for the manifestation of Ajīrṇa are as follows- Factors which vitiates *Agni* are responsible for *Ajīrṇa roga* and the same was described are as follows- Excessive fasting; eating during indigestion; over eating and irregular eating; consumption of unwholesome, heavy, cold, excessively rough and contaminated food; improper administration of purgation, emesis and oleation therapies; emaciation due to disease; faulty adaptation in respect to place, time & season; suppression of natural urges; drinking more quantity of water. Improper sleep, one who eats large quantities of food recklessly like cattle and *Ajīrṇa* is said to be root cause for many diseases.

ईर्ष्याभयक्रोधपरिक्षतेन लुब्धेन रुग्दैन्यनिपीडितेन ।
प्रद्वेषयुक्तेन च सेव्यमानमन्नं न सम्यक् परिणाममेति ॥
S.S.Su. 46/508

Food consumed by person afflicted with jealousy, fear, anger, miseries, grief, helplessness & hatredness does not digest the food properly.

अजीर्ण संप्राप्ति (Samprāpti of Ajīrṇa)

कालेऽपि सात्म्यं लघु चापि भुक्तमन्नं न पाकं भजते नरस्य ॥
S.S.Su. 46/500-501

स दुष्टोऽन्नंः न तत् पचति लघ्वपि ।
अपच्यमानं शुक्तत्वं यात्यन्नं विषरूपताम् ॥
C.S.Ci. 15/44

Due to above etiological factors *Agni* become greatly vitiated and manifest *Ajīrṇa*. Patients of *Ajīrṇa* does not digest the food even though consumed at the proper time, wholesome diet and easily digestible food substances. This undigested food becomes sour in taste and toxic.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi doṣās*
↓
Diminution of *Jaṭharāgni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Localisation of *Vātādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*
↓
Undigested food becomes sour in taste and toxic
↓
Ajīrṇa

अजीर्ण भेद (Ajīrṇa Bheda)

आमं विदग्धं विष्टब्धं कफपित्तानिलैस्त्रिभिः ।
अजीर्णं केचिदिच्छन्ति चतुर्थं रसशेषतः ॥
S.S.Su. 46/499

आमं विदग्धं विष्टब्धं कफपित्तानिलैस्त्रिभिः ।
अजीर्णं केचिदिच्छिन्ति चतुर्थं रसशेषतः ॥
अजीर्णं पञ्चमं केचिन्निर्दोषं दिनपाकि च ।
वदन्ति षष्ठं चाजीर्णं प्राकृतं प्रतिवासरम् ॥
M.Ni. 6/5-6

It is of 6 types namely *Āmājīrṇa, Vidagdh-ājīrṇa, Viṣṭabdh-ājīrṇa* are three varieties of *Ajīrṇa* caused by *Kapha, Pitta* and *Vāta* respectively. Some says *Rasaśeṣājīrṇa* is the fourth variety, fifth variety is *Dinapāki* and sixth variety is *Prākṛta*.

अजीर्ण लक्षण (Ajīrṇa Lakṣaṇa)

तस्य लिङ्गमजीर्णस्य विष्टम्भः सदनं तथा ।
शिरसो रुक् च मूर्च्छा च भ्रमः पृष्ठकटिग्रहः ॥
जृम्भाऽङ्गमर्दस्तृष्णा च ज्वरश्छर्दिः प्रवाहणम् ।
अरोचकोऽविपाकश्च, घोरमन्नविषं च तत् ॥
C.S.Ci. 15/45-66

Common signs and symptoms of Ajīrṇa are as follows : Abdominal distension, general malaise, headache, fainting, giddiness, stiffness in back & waist, yawning, bodyache, thirst, fever, vomiting, tenesmus, anorexia & indigestion. This is a serious condition known as *Annaviṣa.*

विशेष लक्षण (Viśeṣa Lakṣaṇa)

आमाजीर्ण (Āmājīrṇa)

तत्रामे गुरुतोत्क्लेदः शोथो गण्डाक्षिकूटगः ।
उद्गारश्च यथाभुक्तमविदग्धः प्रवर्तते ॥
M.Ni. 6/10

माधुर्यमन्नं गतमामसंज्ञं । *S.S.Su. 46/509*

Food which has attained sweetness is known as *Āmājīrṇa.* It is characterised by heaviness in abdomen, nausea, swelling of the cheeks and eyes & belchings similar to those occurring just after meals.

विदग्धाजीर्ण (Vidagdhājīrṇa)

विदग्धसंज्ञं गतमम्लभावम् । *S.S.Su. 46/509*

विदग्धे भ्रमतृण्मूर्च्छाः पित्ताच्च विविधा रुजः ।
उद्गारश्च सधूमाम्लः स्वेदो दाहश्च जायते ॥
M.Ni. 6/11

Food which has attained sourness & slightly digested is known as *Vidagdhājīrṇa* and it is charcterised by giddiness, thirst, fainting, sour & hot belching, excessive perspiration, burning sensation inside and other symptoms of aggravated *Pitta.*

विष्टब्धाजीर्ण (Viṣṭabdhājīrṇa)

भृशतोदशूलं विष्टब्धमानद्धविरुद्धवातम् ॥
S.S.Su. 46/509

विष्टब्धे शूलमाध्मानं विविधा वातवेदनाः ।
मलवाताप्रवृतिश्च स्तम्भो मोहोऽङ्गपीडनम् ॥
M.Ni. 6/12

Viṣṭabdhājīrṇa is characterised by pricking pain, long stasis, tympanitis, abnormal movement of *Vāta,* obstruction to stool and flatus, delusion, general malaise and other symptoms of *Vāta Vṛddhi.*

रसशेषाजीर्ण (Rasaśeṣājīrṇa)

उद्गारशुद्धावपि भक्तकाङ्क्षा न जायते हृद्गु→ता च यस्य ।
रसावशेषेण तु सप्रसेकं चतुर्थमेतत् प्रवदन्त्यजीर्णम् ॥
S.S.Su. 46/503

रसशेषेऽन्नविद्वेषो हृदयाशुद्धिगौरवे । *M.Ni. 6/13*

Rasaśeṣājīrṇa is characterised by disliking for meals, pure belchings, heaviness and turbidity in the heart, excessive salivation and heaviness.

दिनपाकि अजीर्ण (Dinapāki Ajīrṇa)

अजीर्णं पञ्चमं केचिदित्यादि । निर्दोषम् आध्मानादिदुष्टेरकारकम्। दिनपाकि चेत्यहोरात्रेणाहारः पच्यत इत्युत्सर्गः, यत्र तु मात्रा कालासात्म्यादि दोषादपरदिने पच्यते तद्दिनपाकि ।
M.Ni. 6/5-6 (Madhukoṣa)

The fifth *Ajīrṇa* is *Dinapāki* in which the digestion completes within one day and one night. This occurs due to overages of quantity and timings of food and waiting alone is required for such condition and hence it is not harmful.

प्राकृत अजीर्ण (Prākṛta Ajīrṇa)

प्राकृतं प्रतिवासरमिति प्राकृतमवैकारिकं प्रतिवासरं प्रतिदिनं क्रियमाणम् । अयमभिसन्धिः -अद्यैव भुक्तमन्नं किं जीर्णमजीर्णं वा? न करोतीति पूर्वेभ्यो भिन्नम्।
M.Ni. 6/5-6 (Madhukoṣa)

Prākṛta ajīrṇa prevails till the consumed food is completely digested. After the completion of digestion it manifest symptoms like hunger, thirst, defecation, etc. Clinical features of *Āma* like tympynitis etc are not seen.

अजीर्ण उपद्रव (Ajīrṇa Upadrava)

मूर्च्छा प्रलापो वमथुः प्रसेकः सदनं भ्रमः ।
उपद्रवा भवन्त्येते मरणं चाप्यजीर्णतः ॥
S.S.Su. 46/511

Complications of Ajīrṇa are as follows : Fainting, delirium, vomiting, excess salivation, debility giddiness and death.

Effect of Ajīrṇa

संसृज्यमानं पित्तेन दाहं तृष्णां मुखामयान् ।
जनयत्यम्लपित्तं च पित्तजांश्चापरान् गदान् ॥

यक्ष्मपीनसमेहादीन् कफजान् कफसङ्गतम्।
करोति वातसंसृष्टं वातजांश्च गदान् बहून्॥
मूत्ररोगांश्च मूत्रस्थं कुक्षिरोगान् शकृद्गतम्।
रसादिभिश्च संसृष्टं कुर्याद्रोगान् रसादिजान्॥
C.S.Ci. 15/47-49

When *Ajīrna* combined with *Pitta* causes burning sensation, thirst and diseases of oral cavity. It also manifest *Āmlapitta* and such other *Pittaja Vikarās.*

When *Ajīrṇa* combined with *Kapha* causes *Rājayakṣmā, Pinasa, Prameha* and other diseases causes by *Kapha.*

When *Ajīrṇa* combined with *Vāta* leading to manifestation of various *Vātaja* diseases.

When *Ajīrṇa* afflicts urine, stool and *Dhātus* gives rise to urinary disorders, gastrointestinal diseases and diseases of *Dhātus* respectively.

उपशय/अनुपशय (Upaśaya/Anupaśaya)

दिवा विबुद्धे हृदये जाग्रतः पुण्डरीकवत्।
अन्नमक्लिन्नधातुत्वादजीर्णेऽपि हितं निशि॥
हृदि सम्मीलिते रात्रौ प्रसुप्तस्य विशेषतः।
क्लिन्नविस्त्रस्तधातुत्वादजीर्णे न हितं दिवा॥
S.S.Su. 46/530-531

During day time, the person is awake, his heart is open like the lotus flower and the tissues not soaked with more of moisture, consuming food at night is beneficial though the person is suffering from indigestion. But during night time, the heart being closed because of sleep, and the tissues having more moisture, consuming food at night is not beneficial for the *Ajīrṇa* patients. *Upaśaya* and *Anupaśaya* measures like that of *Grahaṇī Roga.*

आनाह (Ānāha)

Abdominal bloating occurs when the gastrointestinal (GI) tract is filled with air or gas.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

आनाहः मूत्रपुरीषरोधरोगः तत्पर्यायः विबन्धः विष्टम्भः मलरोधनः। *Śabdakalpdruma*

आनाहो वातमूत्रपुरीषादि निरोधः। *S.S.Su. 26/10 (Dalhaṇa)*

Obstruction of urine and faeces or obstruction of flatus, urine, faeces etc. is known as *Ānāha, Vibandha, Viṣṭambha* and *Malarodhajana* are the synonyms of *Ānāha.*

आनाह उदरपूरणलक्षणः स च वातोत्कटदोषैर्जनितः॥
Āḍd.Śa.Prā. 7/49

The fullness of the abdomen due to aggravation of *Vāta* is called *Ānāha.*

The disease which produces the features of *Udarapūrana* (fullness of the abdomen) due to aggravation of *Vāta* is called *Ānāha.*

आनाहाध्मानभेदः॥ *Kā.Śa.Pra. 7/49*

Ācārya Kāśirāma described that the disease *Ānāha* is a type of *Ādhmāna* (distension of abdomen).

आनाहो बद्धोदरताः। *A.H.Ni. 6/146 (Hemādri)*

आनाहो जङ्गराध्मानम्। *A.H.Ni. 28/8 (Aruṇdatta)*

Obstruction in the abdomen or distension of abdomen is known as *Ānāha.*

आनाह्यते आध्मानयुक्तो भवति।
S.S.Utt. 56/7-9 (Dalhaṇa)

Ācārya Dalhaṇa opines that the distension of abdomen due to flatus.

यस्य वातः प्रकुपितः कुक्षिमाश्रित्य तिष्ठति।
नाधो व्रजति नाप्यूर्ध्वमानाहस्तस्य जायते॥
C.S.Su. 18/32

The aggravated *Vāta* that gets localized in the abdomen does not move either upwards or downwards and leads to the disease *Ānāha.*

आमं शकृद्वा निचितं क्रमेण भूयो विबद्धं विगुणानिलेन।
प्रवर्त्तमानं न यथास्वमेनं विकारमानाहमुदाहरन्ति॥
तस्मिन् भवन्त्यामसमुद्भवे तु तृष्णाप्रतिश्यायशिरोविदाहाः।
आमाशये शूलमथो गुरुत्वं हृल्लास उद्गारविघातनञ्च॥
स्तम्भः कटीपृष्ठपुरीषमूत्रे शूलोऽथ मूर्च्छा स शकृद्वमेच्च।
श्वासश्च पक्वाशयजे भवन्ति लिङ्गानि चात्रालसकोद्भवानि॥
S.S.Utt. 56/20-22

Gradual accumulation of *Āma* or stool inside the *Mahāsrotas* and it is obstructed from going out by the excacerbated *Vāta* and is not expelled out through their respective channels is known as *Ānāha.*

If *Ānāha* manifests by *Āma* produces thirst, rhinitis, burning sensation in the head, pain & heaviness in stomach, nausea and obstruction to belchings.

If *Ānāha* caused by stool produces stiffness in waist & back, non elimination of stool & urine, pain in abdomen, fainting, faecal vomiting, dyspnoea and symptoms of *Alasaka.*

आटोप (Āṭopa)

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

आटोपः गुडगुडाशब्दः ॥ *C.S.Ci. 14/21 (Cakrapāṇi)*

आटोपः गुडगुडाशब्दविशेषो वातजन्य ॥
C.S.Ni. 8/6-7 (Cakrapāṇi)

Movement of *Vāta* with gurgling sound in the abdomen is known as *Ātopa.*

आटोपः कुक्षौ शब्दवद्वातभ्रमणम् ।
C.S.In. 5/12-13 (Cakrapāṇi)

Movement of *Vāta* with gurgling or clicking noise in the abdomen is known as *Āṭopa.*

आटोप आध्मानम् । *S.S.Ci. 8/8, S.SNi. 2/8 (Dalhaṇa)*

आटोप उदरस्याध्मानं । *S.S.Su. 46/474 (Dalhaṇa)*

Distension of abdomen is termed as *Āṭopa*

आटोप उदरापुराः । *S.S.Utt. 42/8 (Dalhaṇa)*

Fullness of abdomen is *Āṭopa*

आटोपः सञ्चलनं, तेन सहवर्तते इति साटोपम् ।
S.S.Ni. 1/88 (Gaya)

आटोपश्चलचलमिति । *M.Ni. 22/28 (Madhu)*

Movement of *Vāta* with gurgling sound in the abdomen is known as *Āṭopa.*

आटोपः क्षोभः । *A.H.Su. 28/8 (Aruṇdatta)*

आटोपः आध्मानं क्षोभमित्यन्ते ॥
S.S.Su. 15/15 (Dalhaṇa)

Distension of abdomen with irritation or trembling is known as *Āṭopa.*

आटोपः रुजापूर्वकक्षोभः । *S.S.Ni. 9/20 (Dalhaṇa)*

आटोपः सतोदो वातसंक्षोभः ॥ *SS.Su. 26/10 (Dalhaṇa)*

Irritating pain in the abdomen is known as *Āṭopa.*

आटोपः सतोदो वातसंक्षोभः । *S.S.Su. 26/10 (Dalhaṇa)*

Movement of *Vāta* with pricking pain in the abdomen is known as *Āṭopa.*

आटोपो रुजापूर्वक उदरक्षोभः ।
S.S.Su. 21/32 (Dalhaṇa)

Āṭopa means abnormal movements of *Vāta* with pain.

आध्मान (Ādhmāna)

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

उदर वायोरतीव संचय अनिलपूरणम् । *A.Śa.K.*

Fullness of abdomen with *Vāta* due to accumulation of *Vāta* is known as *Ādhmāna.*

वायुना कुपितेन नाभेरधः सशुलमापूरः आध्मानः ।
A.Sa.Su. 5/8

Pain and filling up of *Vāta* below the umbilicus due to aggravation of *Vāta* is known *as Ādhmāna.*

अनिलश्च रूद्धमार्गतया आध्मान करोतीति ॥
C.S.Si. 7/22 (Cakarpāṇi)

Obstruction for the passage of *Vāta* causes *Ādhmāna.*

उदरस्याध्मानम् वायुना परिपूरणम् । *A.Śa.K.*

आध्मान वातेनोदरपूरणम् । *A.H.Su. 11/13 (Hemādri)*

Fullness of abdomen with *Vāta* is known as *Ādhmāna.*

साटोपमत्युग्ररुजमाध्मातमुदरं भृशम् ।
आध्मानमिति जानीयाद्घोरं वातनिरोधजम् ॥
विमुक्तपार्श्वहृदयं तदेवामाशयोत्थितम् ।
प्रत्याध्मानं विजानीयात् कफव्याकुलितानिलम् ॥
S.S.Ni. 1/88-89

Distension of abdomen associated with sound and severe pain is known as *Ādhmāna* and it manifest due to suppression of *Vāta.*

The same commences from *Āmāśaya* sparing the flanks and region of the heart is known as *Pratyādhmāna* and it manifest by association with *Vāta & Kapha.*

MCQs

1. *Udaraadmana* with *athyugraruja* is the feature of
   a. *Anaha* b. *Ashtila*
   c. *Gulma* d. *Udara*
2. *Gandaakshikootasotha* seen in
   a. Amaajeerna b. Rasaseshaajeerna
   c. Ajeernaupadrava d. Alasaka
3. Types of *Ajeerna* according to *Madava Nidan*
   a. 3 b. 5
   c. 6 d. 7
4. *Amajeerna* leads to
   a. Alasaka b. Vishoochika
   c. Vidagdhaajeerna d. Vishtabdhaajeerna
5. *Shakritvamana* is the *Asadyaakshana* of
   a. Anaha *b. Udavarta*
   c. Rakta pitta *d. Soola*
6. Types of *Anaha*
   a. 4 b. 3
   c. 2 d. 13
7. *Urdhwa-adhovatamargarodha* is seen in
   a. *Ashteela, anaha* b. *Pratyashteela*
   c. *Anaha, Gulma* d. *Adhmana, Gulma*
8. *Dinapaki-ajeerna* is
   a. *Vatika* b. *Kaphaja*
   c. *Tridoshaja* d. *Nirdosha*
9. *Prativasara* nature is seen in
   a. *Vatajaajeerna*
   b. *Kaphajaajeerna*
   c. *Rasa seshaajeerna*
   d. *Prakritaajeerna*
10. The feature of *Rasa seshaajeerna* include all except
    a. *Anna vidwesha* b. *Hridayaasudhi*
    c. *Gourava* d. *Malavataapravrthi*
11. *Alasakalak*shana seen in
    a. *Purishajaanaha* b. *Amajaanaha*
    c. *Adhmana* d. *Atopa*

Answer

1.	a	2.	a	3.	c
4.	b	5.	a	6.	c
7.	a	8.	d	9.	d
10.	d	11.	a		

CHAPTER 2

Chhardi Roga

Introduction

- Vomiting, also known medically as "emesis" and colloquially as throwing up, retching, heaving, hurling, puking, tossing, or being sick, is the forcible voluntary or involuntary emptying of stomach contents through the mouth or, less often, the nose.
- Nausea is an uneasiness of the stomach that often comes before vomiting. Vomiting is the forcible voluntary or involuntary emptying ("throwing up") of stomach contents through the mouth.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

छर्दिः छर्दयत्यनेनेति । वमिरोगाः । *Śabdakalpdruma*

The forcible voluntary or involuntary emptying known as *Chardi* or *Vamirogāḥ*

छर्दिवमनविशेषः । वेगैः कृत्वा आननं छादयतीति छर्दिः ।
Āḍd.Śa.Prā. 7/28

The throwing up, retching, heaving, hurling, puking, tossing, or being sick, is the forcible voluntary or involuntary emptying of stomach contents through the mouth is known as *Chardi*

ऊर्ध्वातिदोषप्रवृत्तिरूपा छर्दिरुच्यते ।
C.S.Ci. 20/1 (Cakarpāṇi)

The elimination of aggravated *doṣas* through upward orifice i.e. mouth is called *Chardi.*

छादयन्नाननं वेगैरर्दयन्नङ्गभञ्जनैः ।
निरुच्यते छर्दिरिति दोषो वक्त्राद्विनिश्चरन ॥
S.S.Utt. 49/6

Forceful discharge of stomach contents with tearing pain in the body is known as *Chardi.*

छर्दि निदान (Chardi Nidāna)

अतिद्रवैरतिस्निग्धैरहृद्यैर्लवणैरति ।
अकाले चातिमात्रैश्च तथाऽसात्म्यैश्च भोजनैः ॥
श्रमात् क्षयात्तथोद्वेगादजीर्णात् कृमिदोषतः ।
नार्य्याश्चापन्नसत्त्वायास्तथाऽतिद्रुतमश्नतः ॥
अत्यन्तामपरीतस्य छर्देर्वै सम्भवो ध्रुवम् ।
बीभत्सैर्हेतुभिश्चान्यैर्द्रुतमुत्क्लेशितो बलात् ॥
S.S.Utt. 49/3-5

Causative factors of Chardi are as follows : One who consumes food which are excessive liquid, excess unctuousness, unpleasant, excessive salt, meals during improper time, excessive quantity of food intake, unwholesome, exertion, depletion of tissues, mental emotions, indigestion, worm infestations, pregnant women, consuming food very quickly, too much accumulation of *Āma,* horrifying causes and sudden bouts of emotions.

छर्दि रोग संप्राप्ति (Samprāpti of Chardi Roga)

दोषानुदीरयन् वृद्धानुदानो व्यानसङ्गतः ।
ऊर्ध्वमागच्छति भृशं विरुद्धाहारसेवनात् ॥
S.S.Utt. 49/7

Due to above etiological factors *Vāta* especially *Udāna* & *Vyāna* gets aggravated and makes upward movement of *Doṣās* resulting into the expulsion of *Doṣās* from mouth.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta* especially *Udāna* & *Vyāna* along with *Pitta* and *Kapha*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Localisation of *Vātādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*
↓
Upward movement and Expulsion of *Doṣās* from mouth
↓
Leading to destruction of *Svara* (Voice)
↓
Chardi

छर्दि रोग सप्रांप्ति घटक (Samprāpti Ghaṭaka of Chardi Roga)

Doṣa : *Vāta (Udāna & Vyāna) Pradhāna*

Tṛdoṣaja
Kapha → Kledaka
Pitta → Pācaka
Vāta → Udāna, Vyāna, Samāna
Manas → Dviṣṭārtha Saṁyogaja Chardi

Duṣya : *Dhātu → Rasa*
: *Upadhātu →*
Śārīraja Mala → Puriṣa
Dhātu Mala → Kapha, Pitta
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Mandāgni, Viṣamāgni*
Āma : *Āgnijanya*
Srotas : *Annavaha*
Srotoduṣṭi: *Vimārgagamana & Saṅga*
Sancārasthāna : *Koṣṭha*
Adhiṣṭhāna : *Āmāśaya*
Udbhavasthāna : *Āmaśayottha, Koṣṭha (C.Su 11/41)*
Svabhāva : *Āśukāri*
Prabhāva : Without complications-curable with complications-not curable
Rogamārga : *Abhyantara*

पूर्वरूप (Pūrvarūpa)

प्रसेको हृदयोत्क्लेशो भक्तस्यानभिनन्दनम्।
पूर्वरूपं मतं छर्द्या यथास्वं च विभावयेत्॥
S.S.Utt. 49/8

तापां हृदुत्क्लेशकफप्रसेकौ द्वेषोऽशने चैव हि पूर्वरुपम्॥
C.S.Ci. 20/6

Premonitory signs & symptoms of Chardi are as follows : Excessive salivation, nausea with uneasy feeling in the cardiac region, dislike for food and the same features may seen in next stage also.

छर्दि भेद (Chardi bheda)

Classification

दोषैः पृथक् त्रिप्रभवा चतुर्थी द्विष्टार्थयोगादपि पञ्चमी स्यात्।
C.S.Ci. 20/6

Chardi is classified into five types namely *Vātaja, Pittaja, Kaphaja, Sannipātaja, Dviṣṭārtha Yogaja Chardi.*

Suśruta included *Krimija Chardi.*

विशेष लक्षण (Viśeṣa Lakṣaṇa)

वातज छर्दि निदान (Vātaja Chardi Nidāna)

व्यायामतीक्ष्णौषधशोकरोगभयोपवासाद्यतिकर्शितस्य।
C.S.Ci. 20/7

Causative factors of Vātaja Chardi are as follows : If person become emaciated due to excessive physical exercise, penetrating drugs, grief, chronic illness, fear and fasting.

वातज छर्दि संप्राप्ति (Vātaja Chardi Samprāpti)

वायुर्महास्त्रोतसि संप्रवृद्ध उत्क्लेश्य दोषांस्तत ऊर्ध्वमस्यन्॥
आमाशयोत्क्लेशकृतां च मर्म प्रपीडयंश्छर्दिमुदींरयेत्तु।
C.S.Ci. 20/7-8

Due to above etiological factors *Vāta* gets aggravated in *Mahāsrotas,* incite the local *Doṣās* and throw them upwards and thus cause vomiting due to gastric irritation also producing discomfort after afflicting vital organs and manifest *Vātaja Chardi.*

वातज छर्दि लक्षण (Vātaja Chardi Lakṣaṇa)

हृत्पार्श्वपीडामुखशोषमूर्धनाभ्यर्तिकासस्वरभेदतोदैः॥
उद्गारशब्दप्रबलं सफेनं विच्छिन्नकृष्णं तनुकं कषायम्।
कृच्छ्रेण चाल्पं महता च वेगेनार्तोऽनिलाच्छर्दयतीह दुःखम्॥
C.S.Ci. 20/8-9

प्रच्छर्दयेत् फेनिलमल्पमल्पं शूलार्दितोऽभ्यर्दितपार्श्वपृष्ठः।
श्रान्तः सघोषं बहुशः कषायं जीर्णेऽधिकं साऽनिलजा वमिस्तु॥
S.S.Utt. 49/9

Vātaja Chardi is characterised by pain in heart and sides of chest, dryness of the mouth, pain in the head & umbilical region, cough, hoarseness of voice, pricking pain; patients vomits with great sound of eructations, which is frothy, scattered, blackish, thin & astringent in taste; patient vomits with severe impulse with difficulty only small amount associated with great distress and vomiting is more after digestion of food.

पित्तज छर्दि निदान (Pittaja Chardi Nidāna)

अजीर्णकट्वम्लविदाह्यशीतै.............................।
C.S.Ci. 20/10

Causative factors of Pittaja Chardi are as follows:- Intake of food during indigestion, consumption of pungent, sour, *Vidāhi* and cold substances in excess.

पित्तज छर्दि सप्रंाप्ति (Pittaja Chardi Samprāpti)

आमाशये पित्तमुदीर्णवेगम् । रसायनीभिर्विसृतं प्रपीड्य मर्मोर्ध्वमागम्य वमिं करोति ॥ *C.S.Ci. 20/10*

Due to etiological factors, *Pitta* situated in *Āmāśaya* gets aggravated and circulates through *Rasāyani* and afflicts the vital organs situated in the upper portion of the body and manifest *Pittaja Chardi.*

पित्तज छर्दि लक्षण (Pittaja Chardi Lakṣana)

मूर्च्छापिपासामुखशोषमूर्धताल्वक्षिसंतापतमोभ्रमार्तः ।
पीतं भृशोष्णं हरितं सतिक्तं धूम्रं च पित्तेन वमेत् सदाहम् ॥
C.S.Ci. 20/11

योऽम्लं भृशं वा कटुतिक्तवक्त्रः पीतं सरक्तं हरितं वमेद्वा ।
सदाहचोषज्वरवक्त्रशोषो मूर्च्छाऽन्वितः पित्तनिमित्तजा सा ॥
S.S.Utt. 49/10

Pittaja Chardi is characterised by fainting, thirst, dryness of mouth, burning sensation in the head, palates & eyes, feeling of darkness, giddiness, patient vomits yellow, excessively hot, green, mild red, bitter and smoky material associated with burning sensation, vomited material is very sour, mouth has pungent & bitter tastes, sucking pain & fever.

कफज छर्दि निदान (Kaphaja Chardi Nidāna)

स्निग्धातिगुर्वामविदाहिभोज्यैः स्वप्नादिभिश्चैव ।
C.S.Ci. 20/12

Causative factors of Kaphaja Chardi are as follows : Excessive intake of unctuous, heavy, uncooked, burning food, excessive sleep or sleep during day time.

कफज छर्दि संप्राप्ति (Kaphaja Chardi Samprāpti)

कफोऽतिवृद्धः। उरः शिरो मर्म रसायनीश्च सर्वाः समावृत्य वमिं करोति ॥ *C.S.Ci. 20/12*

Due to etiological factors *Kapha* gets aggravates and afflicts chest, head, vital organs and *Rasāyanis* to cause *Kaphaja Chardi.*

कफज छर्दि लक्षण (Kaphaja Chardi Lakṣaṇa)

तन्द्रास्यमाधुर्यकफप्रसेकसंतोषनिद्रारुचिगौरवार्ताः ।
स्निग्धं घनं स्वादु कफाद्विशुद्धं सलोमहर्षोऽल्परुजं वमेत्तु ॥
C.S.Ci. 20/13

यो हृष्टरोमा मधुरं प्रभूतं शुक्लं हिमं सान्द्रकफानुविद्धम् ।
अभक्तरुग्गौरवसादयुक्तो वमेद्वमी सा कफकोपजा स्यात् ॥
S.S.Utt. 49/11

Kaphaja Chardi is characterised by drowsiness, sweetishness in mouth, excessive salivations, contentment, sleep, anorexia, heaviness; vomits unctuous, thick, sweet, large quantity, white in colour mixed with thick *Kapha,* cold, free from any undesirable smell; horripitation, pain in empty stomach, debility and slight discomfort.

सन्निपातज छर्दि निदान (Sannipātaja Chardi Nidāna)

समश्नतः सर्वरसान् प्रसक्तमामप्रदोषर्तुविपर्ययैश्च ।
C.S.Ci. 20/14

Causative factors of Sannipātaja Chardi are as follows : Habitual consumption of wholesome & unwholesome together, having all the six tastes, due to *Āma Doṣās* seasonal perversations.

सन्निपातज छर्दि संप्राप्ति (Sannipātaja Chardi Samprāpti)

सर्वे प्रकोपं युगपत् प्रपन्नाश्छर्दिं त्रिदोषां जनयन्ति दोषाः ॥
(C.S.Ci. 20/14)

Due to etiological factors all the three *Doṣās* vitiated simultaneously to cause *Sannipātaja Chardi.*

सन्निपातज छर्दि लक्षण (Sannipātaja Chardi Lakṣaṇa)

शूलाविपाकारुचिदाहतृष्णाश्वासप्रमोहप्रबलाप्रसक्तं ।
छर्दिस्त्रिदोषाल्लवणाम्लनीलसान्द्रोष्णरक्तं वमतां नृणां स्यात्॥
C.S.Ci. 20/15

Sannipātaja Chardi is characterised by colic pain, indigestion, anorexia, burning sensation, thirst, dyspnoea, and fainting which are of serious nature and persistent. Patient vomits saline, sour, blue, viscous, hot & red.

छर्दि उपद्रव (Chardi Upadrava)

Complication of Chardi are as follows:-

विट्स्वेदमूत्राम्बुवहानि वायुः स्त्रोतांसि संरुध्य यदोर्ध्वमेति ।
उत्सन्नदोषस्य समाचितं तं दोषं समुद्धूय नरस्य कोष्ठात्॥
विण्मूत्रयोस्तत् समवर्णगन्धं तृट्श्वासहिक्कार्तियुतं प्रसक्तम् ।
प्रच्छर्दयेद्दुष्टमिहातिवेगात्तयाऽर्दितश्चाशु विनाशमेति ॥
C.S.Ci. 20/16-17

Excessively excacerbated *Vāta* obstruct the channels carrying faeces, sweat, urine and water, moves upwards & expels accumulated *doṣās* out of gastrointestinal tract in the person having excited *doṣās*; vomiting material having the colour & smell of the urine & stool, thirst, dyspnoea, hiccup, foetid and bouts of vomiting with great force. These patients die immediately.

द्विष्टार्थ योगज छर्दि (Dviṣṭārtha Yogaja Chardi)

द्विष्टप्रतीपाशुचिपूत्यमेध्यबीभत्सगन्धाशनदर्शनैश्च ।
यच्छर्दयेत्तप्तमना मनोघ्नैर्द्विष्टार्थसंयोगभवा मता सा ॥
C.S.Ci. 20/18

Smelling, eating and seeing despicable, antagonistic, unclean, putrid, unholy, and gruesome ingredients and objects, afflicts the mind and manifest *Dviṣṭārtha Yogaja Chardi*.

कृमिज छर्दि (Kṛmija Chardi)

शूलहल्लासबहुला कृमिजा च विशेषतः ।
कृमिहृद्रोगतुल्येन लक्षणेन च लक्षिता ॥
S.Utt. 49/14

If *Chardi* manifest due to *Krimi* is known as *Kṛmija* Chardi and it is characterised by pain in abdomen & nausea and other symptoms of *Kṛmija Hṛdroga* are observed.

साध्या साध्यता (Sādhyā Sādhyātā)

क्षीणस्य या छर्दिरतिप्रवृद्धा सोपद्रवा शोणितपूययुक्ता ।
सचन्द्रिकां तां प्रवदन्त्यसाध्या साध्यां चिकित्सेदनुपद्रवां च ॥
C.S.Ci. 20/19

Prognosis of Chardi- Excessive vomiting in emaciated person, which is too severely complicated and associated with blood & pus and moon like lustre is indicative of the incurable nature of disease. Vomiting which is free from complications is curable.

उपशय/पथ्य (Upaśaya/Pathya)

Auṣadha → *Pippalyādi kalka, Kapitthādiyoga, Candanādi Yoga.*

Anna → Foods which are light, well dried, wholesome and *Jvaraghna* decoctions may be used.

Vihāra → Patient should be made to hear pleasing talks, consoled & encouraged, mythological stories, friends & amorous movements, pleasing smell from earth, flower, vinegar & sour fruits.

Since all the different types of vomitting are caused by the affliction of *Āmāśaya* (stomach including small intestines), it is necessary in the first instance, to administer *Laṅghana* (fasting) therapy to the patient. *Laṅghana* should not be advised for *Vātaja chardi* patients. Patient suffering from vomitting may also be given *Samśodhana* (elimination) therapies for the elimination of *Kapha* and *Pitta*. (B.R. 19/1)

The *Pathya* in the case of vomitting includes *Harītakī* (chebulic *Haraḍa*), pomegranate, lemon, *Jātīphala* (nutmeg), *Netrabālā*, fresh leaves of *Neem*, *Vāsaka* leaves, jaggery, *Śatā*, *Nāgakeśara*, *Rāgā*, *Khaḍā*, *Kambalikā*, wine, *Vetāgra*, coriander, coconut, *Āmalakī*, mango, grapes, berry and ripe catechu, etc. The patient should also sprinkle cold water on his face; he should anoint his body with *Kalka* of *Kastūrī* and sandalwood, powder of flowers and other scented materials. Moving around in moonlight is also helpful for the patient of *Chardi*. Administration of the *Dāhakarma* (cauterization) slightly over the naval is also helpful in such cases. The non-veg items useful in the case include the flesh of rabit, peacock, partridge, wood-cutter and the like. (B.R. 19/29-34)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The avoidable (*Apathyas*) in case of *Chardi*: One should avoid in indisposition of *Chardi*, the following: *Nasya* (medication through nose), *Basti* (enema therapy), *Sveda* (sweating), blood-letting, brushing tooth through twigs, liquid meals, beholding disgusting scenes, fear, emotional intensity. And warm, very oily, incompatible, displeasing food items, *Śimbi*, *Bimbi*, bitter gourd, flowers of the *Madhuka* tree and like vegetables, roots of *Citraka* herb, smaller cardamom, mustard and *Devadāli*, etc. are to be avoided in the indisposition. Exercising, holding an umbrella and applying *Añjana*, these are also harmful to the patients. (B.R. 19/35-36).

MCQs

1. Which among the following is not the *asadhyalakshana* of *Chardhi*
   a. *Shonitayukta Chardi*
   b. *Katutiktavaktra*
   c. *Sachandrika*
   d. *Puyayukta Chardi*
2. *Raktavamana* seen in
   a. *Paithikachardhi*
   b. *Agantujachardhi*
   c. *Tridoshajachardhi*
   d. *Kaphajachardhi*
3. *Krimijachardhi* is having the features similar to
   a. *Krimijasiroroga* b. *Krimijahridroga*
   c. *Agantujachardhi* d. *Tridoshajachardhi*
4. Number of *Chardhiupadrava* as per *Madavanidana*
   a. 7 b. 8
   c. 9 d. 5
5. *Dosha* responsible for *Chardi* is
   a. *Prana-Udana* b. *Udana-Vyana*
   c. *Prana-Kapha* d. *Apana-Prana*
6. *Praseka hridayotklesha* is
   a. *Purvarupa of Trishna*
   b. *Purvarupa of Chardhi*
   c. *Rupa of Kaphajatrishna*
   d. *Rupa of Kaphajahridroga*
7. Which among the following not the *upadrava* of *Chardhi*
   a. *Asyamdhurya* b. *Trit*
   c. *Shwasha* d. *Hikka*
8. *Shonithapuyayuktha chardhi observed in*
   a. *Agantuja chardhi lakshana*
   b. *Krimija chardhi lakshana*
   c. *Asadyalakshana of Chardhi*
   d. *Tridoshaja chardhi lakshana*
9. *Bhaktasyanabhinandan and dwesho ashane is the feature of*
   a. *Asadyalakshana of Chardhi*
   b. *Agantuja chardhi lakshana*
   c. *Purvarupa of Chardhi*
   d. *Krimijachardhilakshana*
10. *Daha choshajwara& vaktrashosha are the features observed in*
    a. *Agantuja chardhi lakshana*
    b. *Krimija chardhi lakshana*
    c. *Pittaja chardhi lakshana*
    d. *Vataja chardhi lakshana*
11. *Tandra asyamadhurya kapha prasekha santosha observed in*
    a. *Agantuja chardhi lakshana*
    b. *Kaphaja chardhi lakshana*
    c. *Pittaja chardhi lakshana*
    d. *Vataja chardhi lakshana*

Answer

1.	b	2.	c	3.	b
4.	b	5.	b	6.	b
7.	a	8.	c	9.	c
10.	c	11.	b		

CHAPTER
3

Amlapitta/Āmlapitta

Introduction

Hyperchlorhydria; Increased gastric acidity. Gastritis or Hyper-acidity. Kind of a disease caused by the excessive increase in the sour attribute of *pitta*. It is of two types viz. *Ūrdhvaga* (moving upwardss) and *adhoga* (moving downwards). It is characterized by indigestion, fatigue, nausea, bitter and sour eructation, heaviness, burning sensation in the chest and throat and anorexia.

If both *kapha* and *pitta* are predominant in the pathogenesis of *amla pitta,* then the patient suffers from bitter, sour and pungent eructation, burning sensation in the chest (*hrit*), upper abdomen (*kukṣi*) and throat, fainting, anorexia, vomiting, headache, salivation and sweet taste in the mouth.

Acid reflux can be aggravated by many different things, including lifestyle, medication, diet, pregnancy, weight gain, and certain medical conditions.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

अम्लपित्तं चेति अम्लगुणोद्रिक्तं पित्तम् ॥

C.S.Ci. 15/47 (Cakarpāṇi)

Amlapitta is a condition in which *Pitta* has an excess of sour qualities.

विदाहद्यम्लगुणोद्रिक्तं पित्तमम्लपित्तम् ॥

M.Ni. 51/1 (Madhu)

विदाहद्यम्लगुणोद्रिक्तं पित्तमम्लपित्तमित्युच्यते ॥

Āḍd.Śa.Prā. 7/103

विदग्धं पित्तमक्त्लपित्तमिति ॥ *S.Su. 21/11 (Dalhaṇa)*

Amlapitta is a condition in which the *Pitta* becomes augmented because of excessive of increase of sour qualities.

विदग्धं शुक्ततां याति शुक्तमामाशये स्थितम् ।
तदम्लपित्तमित्याहुर्भूयिष्ठं पित्तदूषणात् ॥

Kā.S.Khi. 16/9

Due to *Vidagdha* (improper digestion), the food becomes *Śuktatā* (sour or acidified) and gets localized in the stomach resulting in *Pittadūṣaṇāt* (vitiation of *Pitta)* and produces the disease *Amlapitta.*

आम्लपित्त निदान (Āmlapitta Nidāna)

विरुद्धदुष्टाम्लविदाहिपित्तप्रकोपिपानान्नभुजो विदग्धम्।
पित्तं स्वहेतूपचितं पुरा यत्तदम्लपित्तं प्रवदन्ति सन्तः ॥

M.Ni. 51/1

विरुद्धाध्यशनाजीर्णादामे चामे च पूरणात् ।
पिष्टान्नानामपक्वानां मद्यानां गोरसस्य च ॥
गुर्वविष्यन्दिभोज्यानां वेगानां धारणस्य च ।
अत्युष्णास्निग्धह्वक्षाम्लद्रवाणामतिसेवनात् ॥
फाणितेक्षुविकाराणां कुलत्थानां च शीलनात् ।
भृष्टधान्यपुलाकानां पृथुकानां तथैव च ॥
भुक्त्वा भुक्त्वा दिवास्वप्नादतिस्नानावगाहनात् ।
अन्तरोदकपानाच्च भुक्तपर्युषिताशनात् ॥

Kā.S.Khi. 16/3-6

Etiological factors of Āmlapitta are as follows : One who consume foods which are incompatible, rotten, sour; food substances which increases burning sensation inside; other factors which aggravates *Pitta;* eating before the digestion of previous meal; indigestion; manifestation of *Āma; Āma* develops at tissue levels; use of ground cereals *(Māṣa)*; improperly processed wines & drinks; eating of heavy & moisture producing foods; suppression of natural urges; excessive use of hot, unctuous, dry, sour & liquid articles, consumptions of *Molasses; Kulattha* & sugarcane preparations; parched cereals; wildly growing rice and *Pṛthuka* (flattened rice obtained from boiled paddy); repeatedly sleeping in day time after eating again and again; over bathing; tub bath; drinking water in between food eating and consumption of stale food.

आम्लपित्त संप्राप्ति (Samprāpti of Āmlapitta)

वातादयः प्रकुप्यन्ति तेषामन्यतमो यदा ।
मन्दीकरोति कायाग्निमग्नौ मार्दवमागते ॥

एतान्येव तथा भूयः सेवामानस्य दुर्मतेः ।
यत्किञ्चिद शितं पीतं देहिनस्तद्धि दह्यति ॥
विदग्धं शुक्ततां यादि शुक्तमामाशये स्थितम् ।
तदम्लपित्तमित्याहुर्भूयिष्ठं पित्तदूषणात् ॥
जन्तोर्यदनुबध्नाति लौल्यादनियतात्मनः ।
अविशुष्के यथा क्षीरं प्रक्षिप्तं दधिभाजने ॥
क्षिप्रमेवाम्लतामेति कूर्चीभावं च गच्छति ।
रसाधातौ तथा व्यम्ले भुक्तं मुक्तं विदह्यते ॥
Kā.S.Khi. 16/7-13

Due to above etiological factors *Vātādi Doṣās* get aggravated leading to mildness of *Agni*. At this time, whatever is eaten by an *ignoramus* person, the same gets burnt, due to improper digestion, food gets acidified inside the stomach. If person doesn't follow rules & regulation of diet even at this stage and indulging unwholesome diet due to greed, the same gets severely vitiated due to pitta is known as *Āmlapitta.*

As the milk poured into undried & uncleaned curd pot, immediately it attains sourness & gets inspissated in a brush form, in the same way repeatedly eaten food gets improperly processed into *Rasadhātu* in case of *Āmlapitta.* When *Ajīrṇa* associates *Pitta* causes *Āmlapitta.*

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Vātādi doṣās*

↓

Diminution of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Localisation of *Vātādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*

↓

Leading to acidification of food inside the stomach.

↓

At this if person doesn't follow rules & regulation of diet
and indulging unwholesome diet due to greed

↓

The same gets severely vitiated and immediately it attains sourness

↓

The repeatedly eaten food gets improperly processed into *Rasadhātu*

↓

Āmlapitta

आम्लपित्त संप्राप्ति घटक (Samprāpti Ghaṭaka of Āmlapitta)

Doṣa : *Pitta Pradhānatridoṣaja*
Vāta → Samāna, Udāna
Pitta → Pācaka
Kapha → Kledaka
Dūṣya : *Dhātu → Rasa*
Upadhātu → Tvacā
Śārīrikamala → Purīṣa
Dhātumala → Kapha, Pitta
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Tīkṣṇāgnī, Mandāgni*
Srotas : *Rasavaha, Annavaha*
Srotoduṣṭi : *Saṅga, Vimārgagamana*
Adhiṣṭhāna : *Āmāśaya*
Udbhavasthāna : *Āmāśayottha*
Sancārasthāna : *Mahāsrotas*
Vyaktasthāna : *Āmāśaya, Kaṇṭa, Hṛdaya*
Svabhāva : *Cirakāri & Āśukāri*
Prabhāva : Newly manifested curable with difficulty & chronic disease curable with difficulty only in few persons.
Rogamārga : *Abhyantara*
Roga bheda : *Ūrdhvaga, Adhoga*

सामान्य लक्षण (Sāmānya Lakṣaṇa)

अविपाकक्लमोत्क्लेशतिक्ताम्लोद्गारगौरवेः ।
हृत्कण्ठदाहारुचिभिश्चाम्लपित्तं वदेद्भिषक् ॥
M.Ni. 51/2

Amlapitta is characterised by indigestion, exhaustion, nausea, sour or bitter eructations, heaviness, burning sensation in the chest & throat and loss of appetite.

अधोग आम्लपित्त लक्षण (Adhoga Āmlapitta Lakṣaṇa)

तृड्दाहमूर्च्छाभ्रममोहकारि प्रयात्यधो वा विविधप्रकारम् ।
हृल्लासकोठ्ठानलसादहर्षस्वेदाङ्गपीतत्वकरं कदाचित् ॥
M.Ni. 51/3

Adhoga Āmlapitta is characterised by thirst, burning sensation, fainting, giddiness, delusion, downward movement of various kinds, occasional oppression in chest, skin rashes, diminished functions of *Agni,* horripilations, perspiration and yellowish discolouration of the body.

ऊर्ध्वग आम्लपित्त (Ūrdhvaga Āmlapitta)

वान्तं हरित्पीतकनीलकृष्णमारक्तरक्ताभमतीव चाम्लम् ।
मांसोदकाभं त्वतिपिच्छिलाच्छं श्लेष्मानुजातं विविधं रसेन ॥
भुक्ते विदग्धे त्वथवाऽप्यभुक्ते करोति तिक्ताम्लवमिं कदाचित् ।
उद्गारमेवंविधमेव कण्ठहृत्कुक्षिदाहं शिरसो रुजं च ॥
करचरणदाहमौष्ण्यं महतीमरुचिं ज्वरं च कफपित्तम् ।
जनयति कण्डूमण्डलपिडकाशतनिचितगात्ररोगचयम् ॥

M.Ni. 51/4-6

Ūrdvaga Āmlapitta is characterised by vomiting, which is green, yellow, blue, black, slightly red or bright red colored sour substances, it resembles like mutton washed water, shiny, thin, followed by *Kapha;* patient vomits during digestion of food or in empty stomach, which is either bitter or sour taste associated with belching of similar nature; burning sensation in the throat, chest and upper abdomen; headache, burning sensation in the palms & soles, feeling of great heat, loss of appetite, *Kapha-Pittaja* fever, skin rashes, itching, surrounded by numerous small vesicles on the skin & other troubles to the body.

Classification of Āmlapitta based on Predominance of Doṣās

सानिलं सानिलकफं सकफं तच्च लक्षयेत् ।
दोषलिङ्गेन मतिमान् भिषङ्मोहकरं हि तत् ॥
कम्पप्रलापमूर्च्छाचिमिचिमिगात्रावसादशूलानि ।
तमसो दर्शनविभ्रमविमोहहर्षाण्यनिलकोपात् ॥
कफनिष्ठीवनगौरवजडतारुचिशीतसादवमिलेपाः ।
दहनबलसादकण्डूनिद्राश्चिह्नं कफानुगते ॥
उभयमिदमेव चिह्नं मारुतकफसंभवे भवत्यम्ले ।
तिक्ताम्लकटुकोद्गारहृत्कुक्षिकण्ठदाहकृत् ॥
भ्रमो मूर्च्छारुचिश्छर्दिरालस्यं च शिरोरुजा ।
प्रसेको मुखमाधुर्यं श्लेष्मपित्तस्य लक्षणम् ॥

M.Ni. 51/8-12

Physician should be able to recognise the symptoms of *Āmlapitta* caused by *Vāta, Vātakapha,* & *Kapha* because they are likely to create confusion to him.

Vātapradhāna Āmlapitta

It is characterised by tremors, delirium, fainting, feeling of pins & needles, general weakness, pain, feeling of darkness in front of the eyes, giddiness, delusion and horripilations.

Kapha Pradhāna Āmlapitta

It is characterised by expectoration of thick phelgum, heaviness, lassitude, loss of appetite, coldishness, general weakness, vomiting, coated tongue, burning sensation, loss of strength, itching and excessive sleep.

Vāta-Kapha Pradhāna Āmlapitta

It is characterised by the appearance of the signs of symptoms of *Vātaja Pradhāna & Kaphaja Pradhāna Āmlapitta.*

Kapha-Pittaja Pradhāna Āmlapitta

It is characterised by eructation having bitter, sour and pungent tastes, burning sensation in chest, upper abdomen & throat, giddiness, fainting, loss of appetite, vomiting, lassitude, headache, salivations and sweetishness in mouth.

साध्यासाध्यता (Sādyāsādhyatā)

रोगोऽयमम्लपित्ताख्यो यत्नात् संसाध्यते नवः ।
चिरोत्थितो भवेद्याप्यः कृच्छ्रसाध्यः स कस्यचित् ॥

M.Ni. 51/7

Prognosis of Āmlapitta- Recently manifested disease responds to treatment with difficulty; chronic *Āmlapitta* is manageable or curable with difficulty in few persons.

उपशय/पथ्य (Upaśaya/pathya)

In cases of *Ūrdhva Āmlapitta* where *Śleṣma* has high concentration one should try *Vamana* therapy. Similarly, in cases of *Adhogata Āmlapitta* where *Pitta* has high concentration one should go with *Virecana* therapy. After carrying out either of these therapies one should administer the *Basti* therapy. Apart from the above measures, one can include the following edibles in their diet: rice of old *Śāli* variety, *Yava*, wheat, *Mudga*, meat and meat soups of wild animals and birds, cool boiled water, sugar, honey, *Saktu*, *Karkoṭa*, *Kāravellaka*, *Paṭola* leaves, *Hilmocika*, *Vetāgra*, well ripened fruit of *Kuṣmāṇḍa*, flowers of banana tree, *Vāstuka*, *Kapittha*, pomegranate, *Āmalakī* and all *Tikta* juices and edibles. One should also drink liquids which are anti-*Kapha* and anti-*Pitta*. (B.R. 56/156-159)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following are strictly prohibited for a patient of *Āmlapitta*: fresh paddy, *Viruddhaśana*, *Pitta* increasing meals, obstructing the flow of vomiting, sesame, *Urada*, *Kulattha*, items fried in oil, milk of sheep, *Kānji*, salt, *Āmla* and *Kaṭu* juices, *Guru* products, curd and liquor. (B.R. 56/160-161)

MCQs

1. Types of *Amlapitta*
   a. 2 b. 3
   c. 5 d. 6
2. Which among the following is not a feature of *Urdwaga-amlapitta*
   a. Mamsodakabhachardhi
   b. Bhuktamvidagdhe
   c. Karacharanadaha
   d. Kotha
3. तिक्ताम्लोद्गारगौरवैः is the feature of
   a. *Amlapitta* b. *Raktapitta*
   c. *Ajeerna* d. *Alasaka*
4. कण्डूमण्डलपिडकाशतनिचितगात्ररोगचयम् is the feature of
   a. *Uttanavatarakta* b. *Mandala kushta*
   c. *Urdwaka Amlapitta* d. *Adhogaamlapitta*
5. The term *Amlapitta* coined by *Caraka* in which chapter
   a. *Sutrasthana 25,Chikitsa sthana 15*
   b. *Chikitsa sthana7,Chikitsasthana 12*
   c. *Sutrasthana 26, Chikitsasthana 13*
   d. *Both a & b*
6. *Which among the following is the types of Amlapitta*
   a. *Urdwaga, Adhoga*
   b. *Urdwaga, Adhoga,Tiryak*
   c. *Sama, Nirama*
   d. *Koshtagata,Sakhagata*

Answer

1.	a	2.	d	3.	a
4.	c	5.	d	6.	a

CHAPTER

4

Shool

Introduction

Colic type of pain in the abdomen is identified as *Śūla*. Stabbing pain in general and colic pain in particular.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

शूल रुजायाम् । शुलति लोक रोगः ॥ *Śabdakalpdruma*

शूलति लोकानिति शूलरोगे ॥ *Śabdakalpdruma*

The term *Śūla* means pain or misery and it is called as disease of mankind.

शूलनिर्घातवेदनाजनकत्वाच्छूलसंज्ञा ।

Āḍd.Śa.Prā. 7/42

Severe or sharp quality of pain is known as *Śūla.*

शूलं निखानितमिवासुखं येन तु वेत्त्यसौ ।

S.S.Utt. 42/67

शूलं निखानितमिव कीलकं निखनितमिवेत्यर्थः ।

S.S.Utt. 42/67 (Dalhaṇa)

Discomfort likes that of as if being pricked by pin or spike is known as *Śūla.*

शूलसम्भवत्वादस्य शूलमिति सज्ज्ञा, शूलनिखातवद् वेदनाजकत्वाच्च । *M.Ni. 26/1 (Madhu)*

The word *Śūla* refers to spike-a weapon. The discomfort or pain felt which is similar to being pierced by spike or being digging in or thrust off is called *Śūla.*

शूलरोपणवत्पीडा यस्याकस्मात्प्रजायते ।
त्रिशूलसंभवं चैनं शूलमाहुः पुराविदः ॥

Y.R. 29/3

Severe pain as if being pierced by an arrow is known as *Śūla* according to ancient scholars because it appeared first due to the trident.

शङ्कस्फोटनवत्तस्य यस्मात्तीव्राश्च वेदनाः ।
शूलासक्तस्य लक्ष्यन्ते तस्माच्छूलमिहोच्यते ॥

S.S.Utt. 42/81

शङ्कुस्फोटनवत्तस्य यस्मात्तीव्र च वेदना ।
शूलासक्तस्य भवति तस्माच्छूलमिहोच्यते ॥

Y.R. 29/4

Severe excruciating pain as if being pierced by a spear or nail is known as *Śūla.*

यतश्च तस्मिन् शूलविद्ध इव व्यथते तीव्रवेदनार्दितः ।
कृच्छ्रोच्छवासस्तस्माच्छूलमित्युच्यते ॥

A.Sa.Ni. 11/32

गुल्मो नाम्ना शूलमित्युच्यते । *E.A.Sa.Ni. 11/32*

Pain as if pierced by a spear associated with excruciating pain and difficulty in breathing is known as *Śūla. Ācārya Indu* described that the *Gulma* is *Śūla* by name.

शूल निदान (Śūla Nidāna)

Consumption of *vātādi* diet and activites causes the development of various *Śūla.* Due to etiological factors *Vatādi doṣās* get aggravated and manifest various *Śūla roga* characterized by pain and burning sensation in the abdomin.

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Vātādi doṣās*

↓

Impairment of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Localisation of *Vātādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*

↓

Leading to development of various kinds of abdominal pain like pain in heart, flanks, back, waist & urinary bladder; burning sensation near the umbilicus, pain near the stomach

↓

Śūla

वातज शूल निदान एवं लक्षण (Vātaja Śūla Nidāna Evam Lakṣaṇa)

व्यायामयानादतिमैथुनाच्च प्रजागराच्छीतजलातिपानात्।
कलायमुद्गाढकिकोरदूषादत्यर्थरुक्षाध्यशनाभिघातात् ॥
कषायतिक्तातिविरूढजान्नविरुद्धवल्लूरकशुष्कशाकात्।
विद्शुक्रमूत्रानिलवेगरोधाच्छो-कोपवासादतिहास्यभाष्यात्॥ वायुः
प्रवृद्धो जनयेद्धि शूलं हृत्पार्श्वपृष्ठत्रिकबस्तिदेशे । जीर्णो प्रदोषे च
घनागमे च शीते च कोपं समुपैति गाढम् ॥ मुहुर्मुहुश्चोपशमप्रकोपी
विड्वातसंस्तम्भनतोदभेदैः । संवेदनाभ्यञ्जनमर्दनाद्यैः
स्निग्धोष्णभोज्यैश्च शमं प्रयाति ॥ *M.Ni. 26/2-5*

वातज शूल निदान (Vātaja Śūla Nidāna)

Etiological factors of Vātaja Śūla are as follows : Excessive physical exercises; riding over vehicles & animals; sexual intercourse; awake at night; drinking cold water, consumption of food substances like peas, green gram, *Āḍaki, Koraduṣa* and others in excess; rough substances; eating food during indigestion; injury; foods which are astringent and bitter; germinated grains; incompatible dietetics; dried fish & leafy vegetables; suppression of the urge for feces, semen, urine, flatus; grief, fasting; excessive speaking & laughing.

वातज शूल संप्राप्ति (Vātaja Śūla Samprāpti)

Etiological factors excacerbates *Vāta* and manifest *Vātaja Śūla.*

वातज शूल लक्षण (Vātaja Śūla Lakṣaṇa)

Vātaja Śūla is characterised by pain in heart, flanks, back, waist & urinary bladder; gets aggravated during or after the digestion of food, evening, cloudy & cold weather; pain increases or decreases of its own accord frequently; there is obstruction to stool & flatus associated with pricking & tearing pain; get relieved by the use of hot fomentation, oil massage, fatty and warm foods & drinks.

पित्तज शूल (Pittaja Śūla)

क्षारातितीक्ष्णोष्णविदाहितैलनिष्पावपिण्याककुलत्थयूषैः ।
कट्वम्लसौवीरसुराविकारैः क्रोधानलायासरविप्रतापैः ॥
प्राम्यातियोगादशनैर्विदग्धैः पित्तं प्रकुप्याशु करोति शूलम् ।
तृणमोहदाहातिकरं हि नाभ्यां संस्वेदमूर्च्छाभ्रमचोषयुक्तम् ॥ मध्यन्दिने
कुप्यति चार्धरात्रे विदाहकाले जलदात्यये च । शीते च शीतैः
समुपैति शान्तिं सुस्वादुशीतैरपि भोजनैश्च ॥ *M.Ni. 26/6-8*

पित्तज शूल निदान (Pittaja Śūla Nidāna)

क्षारातितीक्ष्णोष्णविदाहितैलनिष्पावपिण्याककुलत्थयूषैः ।
कट्वम्लसौवीरसुराविकारैः क्रोधानलाया-सरविप्रतापैः ॥
प्राम्यातियोगादशनैर्विदग्धैः पित्तं प्रकुप्याशु करोति शूलम् ।
तृण्मोहदाहातिकरं हि नाभ्यां संस्वेदमूर्च्छाभ्रमचोषयुक्तम् ॥ मध्यन्दिने
कुप्यति चार्धरात्रे विदाहकाले जलदात्यये च । शीते च शीतैः
समुपैति शान्तिं सुस्वादुशीतैरपि भोजनैश्च ॥ *M.Ni. 26/6-8*

The etiological factors for the manifestation of Pittaja Śūla are as follows : Excessive consumption of food substances which are alkaline, heat producing, very hot, food substances which induce burning during digestion, oily preparations, *Niṣpāva* (cow-pea), oil cake, horsegram soup, food substances which are pungent & sour, fermented preparations like *Sauvirā, Surā* etc., anger, exposure to fire, fatigue, prolonged exposure to hot sun, sexual intercourse and irritating food substances.

पित्तज शूल संप्राप्ति (Pittaja Śūla Samprapti)

Due to above etiological factors *Pitta* aggravates in excess & manifest *Pittaja Śūla.*

पित्तज शूल लक्षण (Pittaja Śūla Lakṣaṇa)

It is characterised by thirst, confusion, burning sensation near the umbilicus, perspiration, fainting, giddiness, localised burning sensation; pain aggravates during mid day or mid night, during digestion of food, rainy season; gets relieved by cold substances like cold drinks, cold exposure etc., sweet foods & drinks which is cold.

कफज शूल निदान (Kaphaja Śūla Nidāna)

Etiological factors of Kaphaja Śūla are as follows : Excessive intake of meat of aquatic & marshy land animals; butter milk & other milk products; mutton; sugarcane products; flour preparations of blackgram, sesamum etc., and other *Kapha* increasing factors.

कफज शूल संप्राप्ति (Kaphaja Śūla Samprāti)

Due to above etiological factors *Kapha* gets aggravated in excess & manifest *Kaphaja Śūla.*

कफज शूल लक्षण (Kaphaja Śūla Lakṣaṇa)

It is characterised by *Nausea,* cough, debility, anorexia, salivation, pain near the stomach, stiffness and loss of movement in the abdomen, heaviness in head, pain increases just after taking food, sunrise, autumn & spring seasons.

सन्निपातज शूल (Sannipātaja Śūla)

सर्वेषु दोषेषु च सर्वलिङ्गं विद्याद्भिषक् सर्वभवं हि शूलम् ।
सुकष्टमेनं विषवज्रकल्पं विवर्जनीयं प्रवदन्ति तज्ज्ञाः ॥
M.Ni. 26/11

It is produced by the combination of all the three *Doṣās* simultaneously and manifest the symptoms of all the three *Doṣās,* symptoms persist continuously all the time, powerful, produce severe discomfort like poison & difficult to treat or it should be rejected for the treatment.

आमज शूल (Āmaja Śūla)

आटोपहृल्लासवमीगुरुत्वस्तैमित्यकानाहकफप्रसेकैः ।
कफस्य लिङ्गेन समानलिङ्गमामोद्भवं शूलमुदाहरन्ति ॥
M.Ni. 26/12

Āmaja Śūla is characterised by gurgling sound in the intestines, *Nausea,* vomiting, heaviness, rigidity & distension of abdomen, salivation and other symptoms as described under *Kaphaja Śūla.*

द्वन्द्वज शूल (Dvandvaja Śūla)

वस्तौ हृत्पार्श्वपृष्ठेषु स शूलः कफवातिकः ।
कुक्षौ हृन्नाभिमध्येषु स शूलः कफपैत्तिक ॥
दाहज्वरकरो घोरो विज्ञेयो वातपैत्तिकः ।
M.Ni. 26/13-14

कफवातज शूल (Kapha-Vātaja Śūla)

Kapha-Vātaja Śūla is charcterised by pain in heart, sides of chest and back.

कफपित्तज शूल (Kapha-Pittaja Śūla)

Kapha-Pittaja Śūla is characterised by pain in abdomen, heart and centre of umbilicus.

वातपित्तज शूल (Vāta-Pittaja Śūla)

Vāta-Pittaja Śūla is characterised by severe pain associated with burning sensation and fever.

साध्यासाध्यता (Sādhyāsādhyatā)

एकदोषोत्थितः साध्यः कृच्छ्रसाध्यो द्विदोषजः ।
सर्वदोषोत्थितो घोरस्त्वसाध्यो भूर्युपद्रवः ॥
M.Ni. 26/14-15

If *Śūla* manifests due to single *Doṣa* then it is curable, that produced by combination of any two *Doṣās* then it is curable with difficulty; that produced by combination of all the three *Doṣās,* severe and associated with complications is incurable.

परिणाम शूल (Pariṇāma Śūla)

परिणाम शूल निदान एवं संप्राप्ति (Pariṇāma Śūla Nidāna Evam Samprāpti)

कफपित्ते समावृत्य शूलकारी भवेद्वली ।
भुक्ते जीर्यति यच्छूलं तदेव परिणामजम् ॥
M.Ni. 26/16

Pariṇāma Śūla manifest due to following etiological factors : Factors which brings aggravation of all the three *Doṣās* by their respective etiological factors resulting in the manifestation of *Pariṇamaśūla.*

परिणाम शूल प्रत्यात्म लक्षण (Pariṇāma Śūla Pratyātma Lakṣana)

Pariṇāma Śūla is characterised by pain just after the digestion of food.

वातज परिणामशूल लक्षण (Vātaja Pariṇāma Śūla Lakṣaṇa)

आध्मानाटोपविण्मूत्रविबन्धारतिवेपनैः ।
स्निग्धोष्णोपशमप्रायं वातिकं तद्वदेद्भिषक् ॥
M.Ni. 26/17-18

Vātaja Pariṇāma Śūla is characterised by distension of abdomen, gurgling sound in the abdomen, non elimination of stool and urine, restlessness, tremors, pain gets relieved by fatty & warm foods.

पित्तज परिणामशूल शूल (Pittaja Pariṇāma Śūla)

तृष्णादाहारतिस्वेदं कद्वम्ललवणोत्तरम्।
शूलं शीतशमप्रायं पैत्तिकं लक्षयेद् बुधः॥
M.Ni. 26/18-19

Pittaja Pariṇāma Śūla is characterised by thirst, burning sensation, restlessness, perspiration, burning sensation, pain aggravates after consuming pungent, sour & salty foods but get relieved after taking cold food & cold measures.

कफज परिणाम शूल (Kaphaja Pariṇāma Śūla)

छर्दिहृल्लासंमोहं स्वल्परुग्दीर्घसन्ततिः।
कटुतिक्तोपशान्तं च तच्च ज्ञेयं कफात्मकम्॥
M.Ni. 26/19

Kaphaja Pariṇāma Śūla is characterised by vomiting, nausea, delusion, mild pain persist for long time, pain gets relieved by pungent & bitter foods.

द्वन्द्वज परिणाम शूल (Dvandvaja Pariṇāma Śūla)

संसृष्टलक्षणं बुद्ध्वा द्विदोषं परिकल्पयेत्॥ *M.Ni. 26/20*

Pariṇāma Śūla manifest due to combination of two *Doṣās* and combined features are observed as per dominancy of *Doṣās*.

त्रिदोषज परिणाम शूल (Tridoṣaja Pariṇāma Śūla)

त्रिदोषजमसाध्यं तु क्षीणमांसबलानलम्। *M.Ni. 26/21*

Tridoṣaja Pariṇāma Śūla manifest due to combination of all the three *Doṣās* & manifest the clinical features of all the three *Doṣās*. If it is associated with emaciation, loss of strength & digestion is said to be incurable.

अन्नद्रव शूल (Annadrava Śūla)

जीर्णे जीर्यत्यजीर्णे वा यच्छूलमुपजायते।
पथ्यापथ्यप्रयोगेण भोजनाभोजनेन च।
न शमं याति नियमात्सोऽन्नद्रव उदाहृतः॥
अन्नद्रवाख्यशूलेषु न तावत्स्वास्थ्यमश्नुते।
वान्तमात्रे जरत्पित्तं शूलमाशु व्यपोहति॥
M.Ni. 26/21-23

Annadrava Śūla is characterised by continuous severe pain and not subsiding either by digestion or after digestion or after taking food or in empty stomach. It does not get relieved by any kind of measures. Some people say patient gets relief after vomiting accumulated *Pitta*.

Differentiate Pariṇāma Śūla and Annadrava Śūla

S.No.	Pariṇāma Śūla	Annadrava Śūla
1	It is characterised by pain just after the digestion of food.	It is characterised by continuous severe pain and not subsiding either by digestion or after digestion or after taking food or in empty stomach
2	• *Vātaja Pariṇāma Śūla* gets relieved by fatty & warm foods. • *Pittaja Pariṇāma Śūla* get relieved after taking cold food & cold measures • *Kaphaja Pariṇāma Śūla* gets relieved by pungent & bitter foods	• It does not get relieved by any kind of measures • Some people say patient gets relief after vomiting accumulated *Pitta*.
3	It is classified into *Vātaja Pariṇāma Śūla, Pittaja Pariṇāma Śūla, Kaphaja Pariṇāma Śūla, Dvandvaja Pariṇāma Śūla & Tridoṣaja Pariṇāma Śūla*	No classification mentioned

उपशय/पथ्य (Upaśaya/Pathya)

Wholesome Diets: To patient suffering from colic pain, steam boiled nuggets prepared out of *Māsa* along with its husk and fried in the *Ghṛtam*, should be served as food. Wheat flour mixed with *Ghṛtam* and jaggery is also useful. It can be given along with sugar and cold milk. Lukewarm *Maṇḍa* (water which is strained out after boiling rice) of

Śāli rice free from *Siktā* (sticky portion) is also useful. *Yava* (barley) cooked in milk and added with *Ghṛtam*, vegetable soup and sugar may also be given. The patient can take boiled milk after taking sugar. He can also take *Saktu* (roasted flour) of *Cāka* along with the soup of leaves of *Paṭola*. (B.R. 30/271-273)

The following measures and eatables are considered to be good for the patient suffering from *Śūla* disorder: *Vamana*, *Svedana*, *Laṅghana* (fasting), putting wicks in *Guda*, *Basti* therapy, sleeping during daytime, *Recana*, easily digestible edibles, one year old rice, fried barley's *Maṇḍa*, warm milk, meat soup of wild animals, *Paṭola*, *Śobhāñjana*, *Kārvellaka*, aubergine, ripe mangoes, raisins, *Kapittha*, black salt, *Priyālāsthi*, leaves of *Śāli Nca Śaka*, *Vāstuka*, *sea salt*, *Sauvarcala* salt, asafoetida, dried ginger, *Viḍa* salt, aniseeds, garlic, clove, oil of *Eraṇḍa*, warm water, juice of *Jambīra* lemon, powder of *Kuṭaja* and light *Kṣārās*. (B.R. 30/279-281)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Prohibitions: The patient suffering from colic pain should avoid eating incompatible foods, remaining awake during nights, unequal meals, exercises, sexual intercourse, alcoholic drinks, salt, pungent food, *Dvidala* (pulses having two cotyledons), suppression of the manifested natural urges, worry and anger. (B.R. 30/282-283)

The patient of *Pariṇāma Śūla* should avoid rice obtained from *Śimbi* variety of paddy, edibles containing cold properties, sour eatables and sesame seeds. He should not take meals before digesting the foods earlier taken. He should also avoid sexual intercourse, walking in sun, waking for long hours, getting angry, indulging in grief and the like. (B.R. 30/284-285).

MCQs

1. Types of *Soola*
   a. 6 b. 8 c. 4 d. 5
2. Intake of *Tilasashkuli* is the *nidana* of
   a. Kaphajasoola b. Paithikasoola
   c. Annadravasoola d. Amajasoola
3. *Sukashtamenam visha vajra kalpam* is the feature of
   a. *Amajasoola* b. *Parinamasoola*
   c. *Tridoshajasoola* d. *Annadravasoola*
4. Types of *Parinamasoola*
   a. *8* b. *9* c. *3* d. *7*
5. *Jeerne jeeryathyajeerne va* is the feature of
   a. *Annadravasoola* b. *Parinamasoola*
   c. *Amajasoola* d. *Tridoshajasoola*
6. *Ardharatre vidaha kale jaladatyaye cha* is the feature of
   a. Vatikasula b. Paithikasoola
   c. Annadravasoola d. Amajasoola
7. *Prognosis of Tridoshajasula*
   a. Sadya b. Yapya
   c. Vivarjaneeya d. Krichratama
8. *Bhukte jeeryati yaccchulam tadeva* ——— *fill in the blanks*
   a. Parinamasoola b. Amajasoola
   c. Tridoshajasoola d. Annadravasoola
9. *Prognosis of Tridoshajaparinamasula*
   a. Sadya b. Yapya
   c. Asadya d. Krichratama
10. *Kaphaja shula features observed* in
    a. Amajasoola b. Tridoshajasoola
    c. Annadravasoola d. Parinamasoola
11. Vantamatre jaratpittam shulamashu vyapohati observed in
    a. Amajasoola b. Tridoshajasoola
    c. Annadravasoola d. Parinamasoola

Answer

1.	b	2.	a	3.	c
4.	d	5.	a	6.	b
7.	c	8.	a	9.	c
10.	a	11.	c		

CHAPTER

5

Atisara and Pravahika

अतिसार (Atisāra)

Introduction

During *Satyayuga*, the sacrificial animals were actually released soon after the recitation of the sacrificial quotations, and these animals were not actually killed during the course of sacrifice. However in the age following the performance of the sacrifice by *Dakṣa Prajāpati,* the sons of *Manu,* like *Nariṣyan, Nābhāga, Ikṣāku, Nṛga* & *Śaryāti* started actually assassinating these sacrificial animals during the performance of *Yajña,* thinking that it was so ordained in the scriptures or being pressed by the animals themselves. Subsequently, it became quite impossible to get other animals in the required number, and *Pṛṣadhra* who was performing a sacrifice for longer duration and started sacrificing cows & bulls when other animals were not available. On seeing this the creatures became shocked and when in this afflicted mental state & consequent loss of agni they ingested the killed cows & bulls, it caused *Atisāra* due to heaviness, hotness, unsuitability & use of inauspicious substance. It can be concluded that *Atisāra* originated initially from the sacrifice performed by *Pṛṣadhra.*

अतिसार हेतु (Atisāra Hetu)

गुर्वतिस्निग्धरूक्षोष्णद्रवस्थूलातिशीतलैः ।
विरुद्धाध्यशनाजीर्णैरसात्म्यै श्चापि भोजनैः ॥
स्नेहाद्यैरतियुक्तैश्च मिथ्यायुक्तैर्विषाद्भयात् ।
शोकाद् दुष्टाम्बुमद्यातिपानात् सात्म्यर्त्तुपर्य्ययात् ॥
जलातिरमणैर्वेगविघातैः कृमिदोषतः ।
नृणां भवत्यतीसारो लक्षणं तस्य वक्ष्यते ॥
S.S.Utt. 40/3-5

The following etiological factors are responsible for the causation of Atisāra : Excessive consumption of foods which are heavy for digestion, very fatty, rough, hot, liquid, hard & cold; incompatible food consumption; excessive quantity; uncooked & unaccustomed; improper or excessive administration of oleation and other therapies; consumption of poison; fear; sorrow; drinking contaminated water & wine in excess; adaptation of unwholesome substances contrary to seasonal regimen; excessive indulgence in water sports; suppression of natural urges and suffering from *Kṛmidoṣa.*

अतिसार संप्राप्ति (Atisāra Samprāpti)

संशम्यापां धातुरन्तःकृशानुं वर्चोमिश्रो मारुतेन प्रणुन्नः ।
वृद्धोऽतीवाधः सरत्येष यस्माद् व्याधिं घोरं तं त्वतीसारमाहुः ॥
S.S.Utt. 40/6

Due to consumption of above etiological factors *Āpa Dhātus'* (body fluids) are enhanced in excess and brings diminution of strength of *Agni,* gets mixed with stool, causes downward movement in *Annavaha srotas* by *Vāta* leading to development of excess watery stool is known as *Atisāra.*

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Pitttādi doṣās* and *Āpa Dhātus*

↓

Diminution of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Agitated *doṣās* mixed with stool

↓

causes downward movement in *Annavaha srotas* by *Vāta*

↓

Atisāra (Excess watery stool)

अतिसार संप्राप्ति घटक (Samprāpti Ghaṭaka of Atisāra)

Doṣa : *Vāta pradhāna tridoṣaja*
Vāta → *Samāna, Apāna*

Pitta → Pācaka
Kapha → Kledaka
Dūṣya : *Dhātu → Rasa, Rakta, Māmsa, Meda*
Upadhātu→
Śārīrika Mala → Mūtra & Puriṣa
Dhātumala → Kapha, Sveda, Pitta
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Mandāgni*
Srotas : *Annavaha Puriṣavaha, Srotas, Udakavaha*
Srotoduṣṭi : *Atipravṛtti, Vimārgagamana*
Adhiṣṭhāna: *Mahāsrotas*
Udbhavasthāna : *Āmāśayottha & Pakvāśayottha*
Sancārasthāna : *Annavaha Srotas, Puriṣavaha*
Vyaktasthāna : *Guda*
Svabhāva : *Āśukāri, Cirakāri*
Prabhāva : With complications → Incurable
Manifested newly without complications → Curable
Roga bheda : 6 types
Roga Mārga : *Abhyantara*

पूर्वरूप (Pūrvarupa)

हृन्नाभिपायूदरकुक्षितोदगात्रावसादानिलसन्निरोधाः ।
विट्सङ्ग आध्मानमथाविपाको भविष्यतस्तस्य पुरःसराणि ॥
S.S.Utt. 40/8-9

Premonitory signs & symptoms of Atisāra are as follows : Pricking type of pain in heart, umbilicus, rectum, lower abdomen and flanks; general malaise; non elimination of flatus, constipation, distension of abdomen and indigestion.

रोग भेद (Roga bheda)

एकैकशः सर्वशश्चापि दोषैः शोकेनान्यः षष्ठ आमेन चोक्तः ।
केचित् प्राहुर्नैकरूपप्रकारं नैवेत्येवं काशिराजस्त्ववोचत् ॥
दोषावस्थास्तस्य नैकप्रकाराः काले काले व्याधितस्योद्भवन्ति ॥
S.S.Utt. 40/7-8

Atisāra is classified into six types i.e. *1. Vātaja, 2. Pittaja, 3. Kaphaja, 4. Sannipātaja, 5. Śokaja & 6. Āmaja;* Some people says it is classified into various types but *Kāśirāja* says it is not right, because it has many different stages of the *Doṣās* and appear at various times during the course of the disease.

According to Caraka

1. Vātaja, 2. Pittaja, 3. Kaphaja, 4. Sannipātaja & 5. Āgantuja (Bhayaja & Śokaja).

Classification of Atisāra

सामं चेत् कुणपं शूलैरुपविशेदरोचकी ।
स घनातिविषाकुष्ठनतदारुवचाः पिबेत् ॥
शकृद्वातमसृक् पित्तं कफं वा योऽतिसार्यते ।
पक्वं, तत्र स्ववर्गीयैर्बस्तिः श्रेष्ठं भिषग्जितम् ॥
C.S.Si. 8/19-20

1. *Āmaja,* 2. *Puriṣaja* 3. *Vātaja,* 4. *Raktaja,* 5. *Pittaja,* 6. *Kaphaja.*

If patient passes stool which is *Āmayukta* associated with cadeveric smell, colic pain, anorexia, then he should be given to drink the decoction of *Ghana, Ativiṣa, Nata, Devadāru & Vacā.*

If the patient passes excessively mature stool, flatus, blood, *Pitta* or *Kapha,* this condition should be treated with enema prepared with appropriate drugs.

षण्णामेषां द्विसंसर्गात् त्रिंशद्भेदा भवन्ति तु ।
केवलैः सह षट्त्रिंशद्विद्यात् सोपद्रवानपि ॥
C.S.Si. 8/21

These six varieties of *Atisāra* are subdivided into thirty more varieties depending on the predominance of one or the other *Doṣa.* If six varieties are combined with them, their number becomes thirty six and are manifested along with their complications.

अतीसारः समासेन द्विधा-सामो निरामकः ॥
सासृङ्निरस्त्रः तत्राद्ये गौरवादप्सु मज्जति ।
शकृद्दुर्गन्धमाटोपविष्टम्भातिप्रसेकिनः ॥
विपरीतो निरामस्तु कफात्पक्वोऽपि मज्जति ।
A.H.Ni. 8/13-14

It is classified briefly into two types i.e. *Sāma*

& *Nirāma* and *Sāsṛk* mixed with blood & *Nirāsṛk* not mixed with blood.

Sāma variety is characterised by heavy stools and which sinks in water associated with foul smell, intestinal gurgling, stasis of undigested food in the stomach, pain in abdomen and excessive salivation.

Nirāma variety is characterised by opposite to *Āma* variety symptoms.

Note : Faeces containing more of *Kapha* also sinks but it is mature.

वातज अतिसार (Vātaja Atisāra)

अथावरकालं वातलस्य वातातपव्यायामातिमात्रनिषेविणो रुक्षात्पप्रमिताशिनस्तीक्ष्णमद्यव्यवायनित्यस्योदावर्तयतश्च वेगान् वायुः प्रकोपमापद्यते । *C.S.Ci. 19/5*

वातज अतिसार हेतु (Vātaja Atisāra Hetu)

Etiological factors of Vātaja Atisāra are as follows : If *Vāta Prakṛti* person adopts the following etiology leads to development of *Vātika Atisāra* by aggravating *Vāta*. Excessive exposure to strong wind, hot sun and physical exercise; consumption of rough food; less intake of food; irregular meals; alcohol; excessive sexual intercourse & suppression of natural urges.

वातज अतिसार संप्राप्ति (Vātaja Atisāra Samprāpti)

पक्ता चोपहन्यन्ते; स वायुः कुपितोऽग्नावुपहते मूत्रस्वेदौ पुरीषाशयमुपहत्य, ताभ्यां पुरीषं द्रवीकृत्य, अतीसाराय प्रकल्पते ।
C.S.Ci. 19/5

Due to above etiological factors *Vāta* gets excacerbated and *Agni* is diminished. At this stage agitated *Vāta* forcefully brings down the *Mūtra* & *Sveda* to the *Puriṣāśaya* and liquifies the stool to cause *Ātisāra.*

वातज अतिसार लक्षण/आमातिसार (Vātaja Atisāra Lakṣaṇa/Āmātisāra)

तस्य रुपाणि विज्जलमामं विप्लुतमवसादि रुक्षं द्रवं सशूलमामगन्धमीषच्छब्दमशब्दं वा विबद्धमूत्रवातमतिसार्यते पुरीषं, वायुश्चान्तःकोष्ठे सशब्दशूलस्तिर्यक् चरति विबद्ध इत्यामातिसारो वातात् । *C.S.Ci. 19/5*

The Signs & symptoms of Vātaja Atisāra during Āma stage are as follows : Stool is slimy mixed with mucus; floats on water or spreads on water; precipitating, rough and liquid nature of stool; diarrhoea associated with colic pain, foul smell of the stool; patient passes stool associated with less sound or no sound; retention of urine and flatus; obstructed *Vāta* moves inside the *Koṣṭha* in haphazard direction and causes pain & gurgling sound.

वातज अतिसार लक्षण/पक्वातिसार (Vātaja Atisāra Lakṣaṇa/ Pakvatisara)

पक्वं वा विबद्धमल्पाल्पं सशब्दं सशूलफेनपिच्छापरिकर्तिकं हृष्टरोमा विनिःश्वसञ् शुष्कमुखः कट्यूरुत्रिकजानुपृष्ठपार्श्वशूली भ्रष्टगुदो मुहुर्मुहुर्विग्रथितमुपवेश्यते पुरीषं वातात्; तमाहुरनुग्रथितमित्येके, वातनुग्रथितवर्चस्त्वात् ॥ *C.S.Ci. 19/5*

The signs & symptoms of Vātaja Atisāra during Pakva stage are as follows : Constipation; passes hard stool in little quantity associated with sound & colic pain; stool is frothy & slimy; cutting pain in the anus; horripilation; increased respiration; dryness of mouth; pain in waist, thigh, sacral region, knee, back and sides of the chest; prolapse of rectum; patient frequently passes scabulous stool *(Grathita).*

It is called *Anugrathita Atisāra* because of stool being scabulous by *Vāta.*

पित्तज अतिसार हेतु (Pittaja Atisāra Hetu)

पित्तलस्य पुनरम्ललवणकटुकक्षारोष्णतीक्ष्णातिमात्रनिषेविणः प्रततान्गिसूर्यसंतापोष्ण-मारुतोपहतगात्रस्य क्रोधेर्ष्याबहुलस्य पित्तं प्रकोपमापद्यते । *C.S.Ci. 19/6*

The following etiological factos manifest Pittaja Atisāra due to aggravation of Pitta : *Pittaja prakṛti* individuals if indulges the following etiological factors i:e regular consumption of sour, saline substances in excess; excessive exposure to strong fire, scorching sun & hot winds; psychologically stressed with anger & jealous.

पित्तज अतिसार संप्राप्ति (Pittaja Atisāra Samprāpti)

तत् प्रकुपितं द्रवत्वादूष्माणमुपहत्य पुरीषाशयविसृतमौष्ण्याद् द्रवत्वात् सरत्वाच्च भित्त्वा पुरीषमतिसाराय प्रकल्पते ।
C.S.Ci. 19/6

Due to above etiological factors *Pitta* gets aggravated and suppresses the functions of *Agni* due to liquidity of *Pitta* and reaches the colon and disintegrates the stool because of heat, liquidity & mobility and causes *Pittaja Atisāra.*

पित्तज अतिसार लक्षण (Pittaja Atisāra Lakṣaṇa)

तस्य रुपाणि- हारिद्रं हरितं नीलं कृष्णं रक्तपित्तोपहितमतिदुर्गन्धमतिसार्यते पुरीषं, तृष्णादाहस्वेदमूर्च्छाशूलब्रध्नसंतापपाकपरीत इति पित्तातिसारः ॥ *C.S.Ci. 19/6*

Signs & Symptoms of Pittaja Atisāra are as follows : Patient passes frequent stool which are either yellow, green, blue or black in colour; stool is associated with *Rakta* and *Pitta* and emits foul smell; thirst; burning sensation; excessive sweating; fainting; colic pain; inflammation & suppuration in anus.

कफज अतिसार हेतु (Kaphaja Atisāra Hetu)

श्लेष्मलस्य तु गुरुमधुरशीतस्निग्धोपसेविनः संपूरकस्याचिन्तयतो दिवास्वप्नपरस्यालसस्य श्लेष्मा प्रकोपमापद्यते ।

C.S.Ci. 19/7

If *Kaphaja Prakṛti* individual indulges following etiological factors leading to aggravation of *Kapha* as a result *Kaphaja Atisāra* manifest.

Consumption of heavy, sweet, cold & unctuous ingredients in excessive quantity; over saturation; free from mental exercises; day sleep & sitting idle.

कफज अतिसार संप्राप्ति (Kaphaja Atisāra Samprāpti)

स स्वभावाद् गुरुमधुरशीतस्निग्धः स्त्रस्तोऽग्निमुपहत्य सौम्यस्वभावात् पुरीषाशयमुपहत्योपक्लेद्य पुरीषमतिसाराय कल्पते ।

C.S.Ci. 19/7

Due to above etiological factors *Kapha* gets aggravated. Excacerbated *Kapha* diminishes the power of *Agni* by its heavy, sweet, cold & unctuous nature and makes downward movement and reaches the colon and further due to watery nature affects the colon with excessive fluid and manifest *Kaphaja Atisāra.*

कफज अतिसार लक्षण (Kaphaja Atisāra Lakṣaṇa)

तस्य रुपाणि स्निग्धं श्वेतं पिच्छिलं तन्तुमदामं गुरु दुर्गन्धं श्लेष्मोपहितमनुबद्धशूलमल्पाल्पमभीक्ष्णमतिसार्यते सप्रवाहिकं, गुरुदरगुदबस्तिवंक्षणदेशः कृतेऽप्यकृतसंज्ञ- सलोमहर्षः सोत्क्लेशो निद्रालस्यपरीतः सदनोऽन्नद्वेषी चेति श्लेष्मातिसारः ॥

C.S.Ci. 19/7

Signs and symptoms of Kaphaja Atisāra are as follows- Patient passes stool which is unctuous, white, slimy, thready, *Āma,* heavy, foetid, mixed with mucus associated with frequent colic pain; patient passes little quantity of stool frequently associated with tenesmus; patient suffers from heaviness in abdomen, anus, pelvis, & groin; has motions without knowledge; horripilation; nausea; excessive sleep; idleness; lassitude and aversion towards food.

सन्निपातज अतिसार हेतु (Sannipātaja Atisāra Hetu)

अतिशीतस्निग्धरुक्षोष्णगुरुखरकठिनविषमविरुद्धासात्म्यभोजनात् कालातीतभोजनाद् यत्किंचिदभ्यवहरणात् प्रदुष्टमद्यपानीयपानादतिमद्यपानादसंशोधनात् प्रतिकर्मणां विषमगमनादनुपचाराज्ज्वलनादित्यपवनसलिलातिसेवनादस्वप्नादतिस्वप्नाद्वेगविधारणादृतुविपर्ययादयथाबलमारम्भाद्भयशोकचित्तोद्वेगातियोगात् कृमिशोषज्वरार्शोविकारातिकर्षणाद्वा व्यापन्नाग्नेस्त्रयो दोषाः प्रकुपिता। *C.S.Ci. 19/8*

The following etiological factors are said to be responsible for the manifestation of *Sannipātaja Atisāra* by simultaneous aggravation of all the three *Doṣās :* Excessive consumption of cold, unctuous, hot, heavy, coarse and hard ingredients; irregular meals; consumption of mutually contradictory food substances; unwholesome food; avoiding intake of food; delayed meals, taking food without considering dietetic rules & regulations; drinking polluted water & wine; drinking of alcohol in excess; not undergoing elimination therapies as per seasons. Inappropriate or non administration of therapeutics; excessive exposure to fire, hot season sun, strong wind and bath etc. in strong force of water; no sleep or excessive sleep; suppression of natural urges; seasonal perversations; excessive physical exercise beyond once own physical strength; excessive exposure to fear, grief and anxiety; excessive emaciation due to *Kṛmi, Śoṣa, Jvara, Arśa* and derangement of *Agni.*

सन्निपातज अतिसार संप्राप्ति एवं लक्षण (Sannipātaja Atisāra Samprāpti evam Lakṣaṇa)

भूय एवाग्निमुपहत्य पक्वाशयमनुप्रविश्यातीसारं सर्वदोषलिङ्गं जनयन्ति ॥ *C.S.Ci. 19/8*

Due to above etiological factors all the three *Doṣās* gets aggravated and brings affliction in *Agni* and reaches *Pakvāśaya* and cause *Sannipātaja Atisāra* with signs & symptoms of all the three *Doṣās*.

सन्निपातज अतिसार लक्षण (Sannipātaja Atisāra Lakṣaṇa)

अपि च शोणितादीन् धातूनतिप्रकृष्टं दूषयन्तो धातुदोषस्वभावकृतानतीसारवर्णानुपदर्शयन्ति । तत्र शोणितादिषु धातुष्वतिप्रदुष्टेषु हारिद्रहरितनीलमाञ्जिष्ठमांसधावनसन्निकाशं रक्तं कृष्णं श्वेतं वराहमेदःसदृशमनुबद्धवेदअनमवेदनं वा समासव्यत्यादुपवेश्यते शकृद् ग्रथितमामं सकृत्, सकृदपि पक्वमनतिक्षीणमांसशोणितबलो मन्दाग्निर्विहतमुखरसश्च, तादृशमातुरं कृच्छ्रसाध्यं बिद्यात् ।

एभिर्वर्णैरतिसार्यमाणं सोपद्रवमातुरमसाध्योऽयमिति प्रत्याचक्षीत, तद्यथा पक्वशोणिताभं यकृतखण्डोपमं मेदोमांसोदकसन्निकाशं दधिघृतमज्जतैलवसाक्षीरवेसवाराभमतिनीलमतिरक्तमतिकृष्णमुदकमिवाच्छं पुनर्मेचकाभमतिस्निग्धं हरितनीलकषायवर्णं कर्बुरमाविलं पिच्छिलं तन्तुमदामं चन्द्रकोपगतमतिकुणपपूतिपूयगन्ध्यामाममत्स्यगन्धि मक्षिकाक्रान्तं कुथितबहुधातुस्त्रावमल्पपुरीषमपुरीषं वाऽतिसार्यमाणं तृष्णादाहज्वरभ्रमतमकहिक्काश्वासानुबन्धमतिवेदनमवेदनं वा स्रस्तपक्वगुदं पतितगुदवलिं मुक्तनालमतिक्षीणबलमांसशोणितं सर्वपर्वास्थिशूलिनमरोचकारतिप्रलापसंमोहपरीतं सहसोपरतविकारमतिसारिणमचिकित्स्यं विद्यात्, इति सन्निपातातिसारः ॥

C.S.Ci. 19/9

Excessively aggravated *Vātādi* three *Doṣās* brings abnormality in *Raktādidhātus* as a result it manifest different coloration of stool; nature of vitiation of *Doṣa & Dhātu* exhibit various colours in stool. If not taken care, further vitiation of *Doṣās & Dhātus* manifest following signs & symptoms- Patient passes stool which is either yellow, green, blue, red like *Mañjiṣṭā,* pink (water in which meat is washed), red, black, white and yellowish (like the colour of pig-fat) painful abdomen or painless.

The above mentioned signs & symptoms manifest in their entirety or only few may be observed. Patient passes stool which is scabulous, *Āma,* or may be free from mucus; diminution of *Māmsa, Rakta* and *Bala;* suppression of the power of *Agni;* distaste in mouth. Above symptomatology indicates that the disease is curable with difficulty.

The following signs and symptoms indicates the incurable nature of the disease- Patient passes stool appears like cooked blood, liver pieces, fat, meat water, curd, ghee, marrow, oil, muscle fat, milk and *Vesavāra* (a spicy preparations); patient passes stool which is blue, red, black, transparent like water or tar coloured, greasy; colour of the stool appears like green, blue and brown colours; stool appears like variegated in colour *(Karbura)*, dirty, slimy, fibrous, mixed with mucus and spotted with *Candraka* (moony lustre), cadaverous smell, foetid, putrid, smells like undigested products or like raw fish, attracts flies, stool contain profuse discharge of increased tissues, passing little or no stool associated with thirst, burning sensation, fever, giddiness, fainting, hiccup, asthma excessive pain or no pain, prolapse & suppuration of anus, sphincters come out of their sites or whole of the rectum comes out; excessive loss of strength, *Māmsa & Rakta;* pain in all the joints & bones; patient suffers from anorexia, dislike for everything, delirium & unconsciousness and sudden cessation of the signs & symptoms of the disease.

सन्निपातज अतिसार (Sannipātaja Atisāra)

तन्द्रायुक्तो मोहसादाम्यशोषी वर्चः कुर्यान्नैकवर्णं तृषार्त्तः ।
सर्वोद्भूते सर्वलिङ्गोपपत्तिः कृच्छश्चायं बालवृद्धेष्वसाध्यः ॥

S.S.Utt. 40/13

Sannipātaja Atisāra is characterised by stupor, delusion, debility, dryness of mouth, eliminates faeces of many colours, thirst and has the appearance of specific symptoms of all the *Doṣās.*

It is very difficult to treat and incurable in young & old persons.

आगन्तुज अतिसार (Āgantuja Atisāra)

आगन्तू द्वावतीसारौ मानसौ भयशोकजौ ।
तत्तयोर्लक्षणं वायोर्यदतीसारलक्षणम् ॥

C.S.Ci. 19/11

The exogenous (*Āgantuja Atisāra)* manifest due to mental factors, and it is of two types i.e. *Bhayaja & Śokaja.* Their signs & symptoms are similar to those of *Vātaja Atisāra.*

शोकज अतिसार (Śokaja Atisāra)

तैस्तैर्भावैः शोचतोऽल्पाशनस्य बाष्पावेगः पक्तिमाविध्य जन्तोः ।
कोष्ठं गत्वा क्षोभयत्यस्य रक्तं तच्चाधस्तात् काकणन्तीप्रकाशम् ॥
वर्चोमिश्रं निःपुरीषं सगन्धं निर्गन्धं वा सार्यते तेन कृच्छ्रात् ।
शोकोत्पन्नो दुश्चिकित्स्योऽतिमात्रं रोगो वैद्यैः कष्ट एष प्रदिष्टः ॥
S.S.Utt. 40/14-15

Person who is suffering from various kinds of grief eat less quantity of food, the force of the tears (grief) vitiates the digestive power of man and reaches the gastrointestinal tract, vitiates his *Rakta;* begins to move in the downward direction and passes stool mixed with *Rakta;* the blood resembles *Kākaṇanti* in colour either mixed with stool or without stool comes out from *anus* associated with either bad smell or without smell & passes with difficulty. It is very difficult to treat says the physicians.

आमज अतिसार (Āmaja Atisāra)

आमाजीर्णोपद्रुताः क्षोभयन्तः कोष्ठं दोषा सम्प्रदुष्टाः सभक्तम् ।
नानावर्णं नैकशः सारयन्ति कृच्छ्राज्जन्तोः षष्ठमेनं वदन्ति ॥
S.S.Utt. 40/15-16

Due to *Amājirṇa, Doṣās* excacerbates in excess in abdomen gives rise to elimination of faeces of various colours, passes stool with difficulty and it is difficult to treat.

रक्तातिसार (Raktātisāra)

पित्तातिसारी यस्त्वेतां क्रियां मुक्त्वा निषेवते ।
पित्तलान्यन्नपानानि तस्य पित्तं महाबलम् ॥
कुर्याद्रक्तातिसारं तु रक्तमाशु प्रदूषयेत् ।
तृष्णां शूलं विदाहं च गुदपाकं च दारुणम् ॥
C.S.Ci. 19/69-70

पित्तातिसारी यो मर्त्यः पित्तलान्यतिषेवते ।
पित्तं प्रदुष्टं तस्याशु रक्तातीसारमावहेत् ॥
ज्वरं शूलं तृषां दाहं गुदपाकञ्च दारुणम् ॥
S.S.Utt. 40/116-117

One who is suffering from *Pittaja Atisāra* and does not follow the treatment of *Atisāra* and indulges foods & drinks which cause further aggravation of *Pitta* upto great extent as a result *Rakta* gets vitiated and manifest *Raktātisāra.* It is very difficult for management because it manifest serious condition and patient suffers from thirst, colic pain, burning sensation & inflammation of the ano-rectal region.

Raktātisāra may manifest as a complication of *Pittātisāra* and it may also manifest independently without *Pittātisāra* if person adopts *Pitta* provocative etiological factors in excess.

उपद्रव (Upadrava)

All the *Lakṣaṇa* mentioned under *Sannipātaja Atisāra.*

शूलप्रवाहिकाध्मानपरिकर्त्यरुचिज्वरान् ।
तृष्णोष्णदाहमूर्च्छादींश्चैषां विद्यादुपद्रवान् ॥
C.S.Si. 8/22

Complications of *Atisāra* are colic pain, gripping pain, flatulence, thirst, feeling of hotness, burning sensation, fainting etc.

साध्यासाध्यता एवं अरिष्ठ (Sādhyāsādhyatā evam Ariṣṭa)

सर्पिर्मेदोवेसवाराम्बुतैलमज्जाक्षीरक्षौद्ररूपं स्त्रवेद् यत् ।
मञ्जिष्ठाऽऽभं मस्तुलुङ्गोपमं वा विस्त्रं शीत प्रेतगन्ध्यञ्जनाभम् ॥
राजीमद् वा चन्द्रकैः सन्ततं वा पूयप्रख्यं कर्दमाभं तथोष्णम् ।
हन्यादेतद् यत् प्रतीपं भवेच्च क्षीणं हन्युश्चोपसर्गाः प्रभूताः ॥
असंवृतगुदं क्षीणं दुराध्मातमुपद्रुतम् ।
गुदे पक्वे गतोष्माणमतीसारकिणं त्यजेत् ॥
S.S.Utt. 40/19-21

Stool resembling ghee, fat, water of meat soup, oil, bone marrow, milk, honey, *Mañjiṣṭā* or *Mastuluṅga*, putrid smell, cold, cadaveric smell, appears like *Añjana (Sauvirāñjana),* has separating lines, glistening particles, resembling like pus or slush and hot, these features greatly enhanced and acting as complications and kill the weak persons. The patient whose anal orifice does not close at all, who is emaciated, having severe flatulence, inflammation in the anus, and coldish touch should be rejected.

अतिसार निवृत्ति लक्षण (Atisāra Nivṛtti Lakṣaṇa)

यस्योच्चारं विना मूत्रं सम्यग्वायुश्च गच्छति ।
दीप्ताग्नेर्लघुकोष्ठस्य स्थितस्तस्योदरामयः ॥
S.S.Utt. 40/162

Proper elimination of urine, flatus and stool, enhancement of digestive fire and feeling of lightness in gastro-intestinal tract indicates that patient is recovered from *Atisāra*.

उपशय/पथ्य (Upaśaya/Pathya)

Auṣadha

Kāliñgakādi yoga, Nāgarādi Yoga, Mustādi Yoga, Pippalyādi Yoga, Cāṅgeri ghṛta, Cavyādi ghṛta, Dāryādi ghṛta, Kapitthāṣṭaka Cūrṇa, Dāḍimāṣṭaka Cūrṇa.

Anna

In appropriate time, when patient feels hungry, he should be given light food to eat. This helps the patient to regain appetite, his *Agni* get stimulated and strength is promoted immediately.

In the initial stage, depending upon the wholesomeness, patient should take light food along with butter milk, *Avanti-soma* or *Kāñji* (sour drink), *Yavāgu* (thick gruel), *Tarpaṇa* (roasted flour of cerelas mixed with water), wine, honey, then gradually, the patient should gradually be given *Yavāgu* (thick gruel), *Vilepī* (a sticky gruel), *Khaḍa* (a sour appetiser) *Yūṣa* (vegetable soup) and boiled rice mixed with meat soup, which are prepared by adding digestive stimulants and astringent drugs.

Vihāra

Vāta gets aggravated soon, even by fear and grief, that's why *Vāta* mitigating factors are beneficial and it should be done by providing pleasure & courage to the patient as per patient need.

If the patient of *Atisāra* is hungry, he should be given light food to eat. *Peya* (thin gruel without containg any grains in it) prepared by boiling with appropriate drugs, or fried paddy or roasted-corn flour mixed along with water is also useful for him. The *Maṇḍa* (that does not contain *Siktaka*) can also be taken by the patient. (B.R. 7/11-12)

Pathya (Meals to be taken) for a patient of *Atisāra* and *Pravāhīkā*: Vomiting, *Laṅgana*, sleeping are good for the patient of *Atisāra*. The following edibles and meals are helpful for the patient: old *Śāli* variety of paddy, *Ṣaṣṭhī* variety of rice, *Vilepi* (watery rice), *Maṇḍa* of poppy, soups of lentil and pigeon pea, meatsoup of rabbit, *Aiṇ* (a kind of black deer), *Lāva* bird, deer, *Kapiñjala* (partridge), all kinds of small fishes, the *Śṛīngī* fish, *Madhurālikā* (a kind of fish), medicinal oil, *Ghṛtam* and milk obtained from goats and cow, buttermilk, butter, fresh banana fruits, honey (obtained from smaller bees), *Jambūphalā*, dried ginger, common ginger, *Kamalakanda*, *Kapittha*, *Vikañṭaka*, *Bakula*, *Bilva*, *Tenduka*, *Dāḍima* (both ripe and unripe ones), palm fruits, *Kañcaṭa* leaves, *Cāngerī*, *Vijayā*, *Mañjiṣṭhā*, *Jātiphala*, *Hṛbera*, cumin seeds, *Girimallikā*, *Kustumburu*, *Mahānimba*, all kinds of stomachiac foods, light meals and drinks. The point about two fingers below the naval as well as the point around bottom of backbone, both should be heated in a crescent shape. This measure also helps one to get cure of *Atisāra*. (B.R. 7/183-189)

प्रवाहिका (Pravāhikā)

वायुः प्रवृद्धो निचितं बलासं नुदत्यधस्तादहिताशनस्य ।
प्रवाहमाणस्य मुहुर्मलात्तं प्रवाहिकां तां प्रवदन्ति तज्ज्ञाः ॥
S.S.Utt. 40/138

One who consumes unwholesome foods & drinks leading to aggravation of *Vāta* and pushes the *Kapha* downwards again & again as a result *Kapha* comes out along with stool is known as *Pravāhikā*.

प्रवाहिका भेद एवं लक्षण (Pravāhikā Bheda Evam Lakṣaṇa)

प्रवाहिका वातकृता सशूला पित्तात् सदाहा सकफा कफाच्च ।
सशोणिता शोणितसम्भवा तु ताः स्नेहरूक्षप्रभवा मतास्तु ॥
तासामतीसारवदादिशेच्च लिङ्गं क्रमं चामविपक्वताञ्च ॥
S.S.Utt. 40/140

1. Vātaja, 2. Pittaja, 3. Kaphaja and *4. Raktaja Pravākrikā* are described along with their symptomatology & these may be considered as classification of *Pravāhikā.*

Pravāhika caused by excacerbated *Vāta* produces pain, that caused by *Pitta* produces burning sensation, that caused by *Kapha* produces elimination of stool mixed with *Kapha* and that caused by *Rakta* produces stool mixed with *Rakta.* These kind of *Pravāhika* are produced by more of unctuousness & roughness in *Pakvāśaya.* Stages of *Āma* & *Pakva* and the management principles are similar to that of *Atisāra.*

Differentiate between Atisāra and Pravāhikā

S.No.	Atisāra	Pravāhikā
1	Frequent passing of excessive watery stool	Evacuation of stool mixed with Kapha
2	No strain while evacuating stool	Strain while evacuating stool

Differentiate doṣaja Atisāra

S.No	Vātaja	Pittaja	Kaphuja	Sannipātaja
Etiology	If *Vāta prakṛti* person adopts the following etiology leads to development of *Vātika Atisāra* by aggravating *Vāta*. Excessive exposure to strong wind, hot sun and physical exercise; consumption of rough food; less intake of food; irregular meals; alcohol; excessive sexual intercourse & suppression of natural urges.	*Pittaja prakṛti* individuals if indulges the following etiological factors i:e regular consumption of sour, saline substances in excess; excessive exposure to strong fire, scorching sun & hot winds; psychologically stressed with anger & jealous.	If *Kaphaja Prakṛti* individual indulges following etiological factors leading to aggravation of *Kapha* as a result *Kaphaja Atisāra* manifest. Consumption of heavy, sweet, cold & unctuous ingredients in excessive quantity; over saturation; free from mental exercises; day sleep & sitting idle.	The following etiological factors are said to be responsible for the manifestation of *Sannipātaja Atisāra* by simultaneous aggravation of all the three *Doṣās* : Excessive consumption of cold, unctuous, hot, heavy, coarse and hard ingredients; irregular meals; consumption of mutually contradictory food substances; unwholesome food; avoiding intake of food; delayed meals, taking food without considering dietetic rules & regulations; drinking polluted water & wine; drinking of alcohol in excess; not undergoing elimination therapies as per seasons. Inappropriate or non administration of therapeutics; excessive exposure to fire, hot season sun, strong wind and bath etc. in strong force of water; no sleep or excessive sleep; suppression of natural urges; seasonal perversations; excessive physical exercise beyond once own physical strength; excessive exposure to

				fear, grief and anxiety; excessive emaciation due to *Kṛmi, Śoṣa, Jvara, Arśa* and derangement of *Agni.*
Clinical Feature	Constipation; passes hard stool in little quantity associated with sound & colic pain; stool is frothy & slimy. Patient frequently passes scabulous stool *(Grathita).*	Patient passes frequent stool which are either yellow, green, blue or black in colour; stool is associated with *Rakta* and *Pitta* and emits foul smell	Patient passes stool which is unctuous, white, slimy, thready, *Āma,* heavy, foetid, mixed with mucus associated with frequent colic pain; patient passes little quantity of stool frequently associated with tenesmus;	Excessively aggravated *Vātādi* three *Doṣās* brings abnormality in *Raktādidhātus* as a result it manifest different coloration of stool; nature of vitiation of *Doṣa & Dhātu* exhibit various colours in stool Patient passes stool which is either yellow, green, blue, red like *Mañjiṣṭā,* pink (water inwhich meat is washed), red, black, white and yellowish (like the colour of pig-fat) painful abdomen or painless.
Clinical Feature	patient suffers from pain in waist, thigh, sacral region, knee, back and sides of the chest;	patient suffers from thirst; burning sensation; excessive sweating; fainting; colic pain	patient suffers from heaviness in abdomen, anus, pelvis, & groin;	patient suffers from excessive loss of strength, *Māmsa & Rakta;* pain in all the joints & bones;
Complication	patients of *Vātika Atisāra* may not develop *Raktātisāra* as a complication	patients of *Pittātisāra* may develop *Raktātisāra* as a complication	patients of *Kaphaja Atisāra* may not develop *Raktātisāra* as a complication	

MCQs

1. *'Visramsi'* according to *Acharya Bhoja* is
   - a. Rakta-Pitta b. Atisara
   - c. Pravahika d. Grahani
2. Type of *Atisara* not mentioned by *Susruta*
   - a. Bhayaja b. Sokaja
   - c. Tridoshaja d. Kaphaja
3. Types of *Atisara* according to *Sharangadhara*
   - a. 6 b. 7
   - c. 5 d. 4
4. *Bhrastaguda muhurmuhur vigrathitha features observed in*
   - a. *Vatika Atisara* b. *Paithika Atisara*
   - c. *Tridoshaja Atisara* d. *Sokaja Atisara*
5. *Krite-apyakritasamjna* sa lomaharsha is the feature of
   - a. *Pravahika* b. *Tridoshaja Atisara*
   - c. *Kaphaja Atisara* d. *Bhayaja Atisara*
6. *Nana varna naikashah sarayanthi features observed in*
   - a. *Pittaja Atisara* b. *Tridoshaja Atisara*
   - c. *Shokaja Atisara* d. *Amaja Atisara*

7. Types of *Pravahika* as per *Madavanidana*
   a. 5 b. 4
   c. 3 d. 2

8. विज्जलभामं विप्लुतमवसादि features observed in
   a. Rakta-Pitta b. Vataja Atisara
   c. Pitttaja Atisara d. Grahani

9. According to *Ashtanga Hridaya* the types of *Atisara* include all except
   a. Bhayaja b. Shokaja
   c. Amaja d. Both a & c

10. *Purvarupa of Atisara include all except*
    a. Gatrasada b. Malagraha
    c. Adhmana d. Hrillasa

11. *Kshipramushnamdravamplavam is the feature of which atisara*
    a. Paittika b. Bhayaja
    c. Shokaja d. Both b & c

Answer

1.	c	2.	a	3.	b
4.	a	5.	c	6.	d
7.	b	8.	b	9.	c
10.	d	11.	d		

CHAPTER

6

Grahani

Introduction

Grahaṇi Doṣa refer to diseases located in *Grahaṇī*. This specific term refers to 4 types of *Grahaṇī roga*. This also includes *Agnimāndhya*, *Ajīrṇa* etc. these diseases manifest due to vitiation of *Grahaṇi*. The term *Grahaṇī* as a disease entity specifically used for the ailment *Grahaṇīgada*, which manifest due to malfunctioning of *Grahaṇī*. *(C.S.Ci. 15/38-41 (Cakrapāṇi)*

Other ailments, like *Agnimāndhya* having only their location are not implied by the term *Grahaṇīgada*. In *Atiśāra*, undigested food particles are released but it is not called *Grahaṇī Doṣa* because of difference in its pathogenesis & line of treatment.

Pittadharakalā situated between *Pakvāśaya* & *Āmāśaya* is known as *Grahaṇī*, strength of *Grahaṇī* is *Agni and Agni* resides in the *Grahaṇī;* that's why when *Agni* is vitiated, *Grahaṇī* also become vitiated *(S.S.Utt. 40/169-170).*

निरुक्ति एवं परिभापा (Nirūkti and Paribhāṣā)

ग्रहणीरोगमिति ग्रहण्या रोगो ग्रहणीरोगः ॥

M.Ni. 4/3 (Madhu)

The disease manifest in the *Grahaṇī* is known as *Grahaṇīroga*.

ग्रहणीमाश्रितोऽग्निदोपो ग्रहणी दोषः ॥

C.SCi. 15/1-2 (Cakarpāṇi)

The morbidity of *Agni* located in the *Grahaṇī* is called *Grahaṇī doṣa*.

अग्न्यधिष्ठानमन्नस्य ग्रहणाद्ग्रहणी मता ।

C.S.Ci. 15/56

अग्न्यधिष्ठानमन्नस्य ग्रहणाद्ग्रहणी मता इति ॥

Āḍd.Śa.Prā. 7/8

Grahaṇī is a dwelling place of bio-digestive fire & receives the food is termed as *Grahaṇī*.

अधस्तु पक्वमामं वा प्रवृत्तं ग्रहणीगदः ।

C.S.Ci. 15/51-52

A slightly digested and undigested food substances move downwards is known as *Grahaṇīgada.*

ग्रहणी रोग निदान (Grahaṇī Roga Nidāna)

दुष्यति ग्रहणी जन्तोरग्निसादनहेतुभिः ॥
अतिसारे निवृत्तेऽपि मन्दाग्नेरहिताशिनः ।
भूयः सन्दूपितो वह्निर्ग्रहणीमभिदूपयेत् ॥

S.S.Utt. 40/166-167

अतीसारेपु यो नातियत्नवान् ग्रहणीगदः ।
तस्य स्यादग्निविध्वंसकैरन्यस्य सेवितैः ।

A.H.Ni. 8/15-16

Diseases of *Grahaṇī* manifest due to disordered functions of *Agni*. After the cure of *Atisāra* or who is suffering from mild digestive fire, if they consume unwholesome dietetic regimen leading to further vitiation of *Agni* along with *Grahaṇī*.

ग्रहणी रोग भेद (Grahaṇī Roga Bheda)

Classification of Grahaṇī Roga

एकशः सर्वशश्चैव दोषैरत्यर्थमुच्छ्रितैः ।
सा दुष्टा बहुशो भुक्तमाममेव विमुञ्चति ॥

S.S.Utt. 40/171

वातात् पित्तात् कफाच्च स्यात्तद्रोगस्त्रिभ्य एव च ।
हेतुं लिङ्गं चिकित्सां च शृणु तस्य पृथक् पृथक् ॥

C.S.Ci. 15/58

There are four varieties of *Grahaṇī roga* namely *Vātaja, Pittaja, Kaphaja & Sannipātaja* are described.

The word '*Grahaṇī Doṣa*' includes the following diseases *C.S.Ci. 15/71,C.S.Ci. 15/38-41 (Cakrapāṇi)*

A) *Grahaṇi roga or Grahaṇī gada*
B) *Agnimāndhya*
C) *Ajīrṇa*
D) *Mandāgni*
E) *Tīkṣṇāgni*
F) *Viṣamāgni etc.*

पूर्वरूप (Pūrvarupa)

पूर्वरुपं तु तस्येदं तृष्णाऽऽलस्यं बलक्षयः ।
विदाहोऽन्नस्य पाकश्च चिरात् कायस्य गौरवम् ॥
C.S.Ci. 15/55

तस्योत्पत्तौ विदाहोऽन्ने सदनालस्यतृद्क्लमाः ।
बलक्षयोऽरुचिः कासः कर्णक्ष्वेडोऽन्त्रकूजनम् ॥
S.S.Utt. 40/173

Premonitory signs & symptoms of *Grahaṇī roga* are as follows- Thirst, laziness, diminution of strength, burning sensation during digestion of food, delayed digestion, heaviness in body, general malaise, exhaustion, anorexia, cough, ringing inside the ears and gurgling noise in the abdomen.

ग्रहणी रोग संप्राप्ति (Samprāpti of Grahaṇī Roga)

सा दुष्टा बहुशो भुक्तमाममेव विमुञ्चति ॥ पक्वं वा ॥
S.S.Utt. 40/171-172

दुर्बलो विदहत्यन्नं तद्यात्यूर्ध्वमधोऽपि वा ॥
अधस्तु पक्वमामं वा प्रवृत्तं ग्रहणीगदः ।
उच्यते सर्वमेवान्न प्रायो ह्यस्य विदह्यते ॥
C.S.Ci. 15/51-52

दुर्बलाग्निबला दुष्टा त्वाममेव विमुञ्चति ॥
C.S.Ci. 15/57

Due to etiological factors both *Agni* and *Grahaṇī* becomes disturbed leading to development of *Grahaṇī roga.*

Impaired *Agni* burns the food incompletely which goes either upward or downwards when it moves downwards either in ripe or unripe condition is known as *Grahaṇī gada.* Such patients suffers from sour eructation, burning sensation, thirst along with difficulty in digestion.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi doṣās*
↓
Disturbance of *Agni* and *Grahaṇī*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Localisation of *Vātādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*
↓
Leading to incomplete digestion of food
↓
Which goes either upward or downwards
↓
When it moves downwards either in ripe or unripe condition
↓
Grahaṇī gada.

(Patients suffers from sour eructation, burning sensation, thirst along with difficulty in digestion)

ग्रहणी रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Grahaṇī Roga)

Doṣa : *Pitta Pradhāna Tṛdoṣaja*
Vāta → Samāna
Pitta → Pācaka
Kapha → Kledaka
Dūṣya : *Dhātu → Rasa*
Upadhātu →
Śārīrika mala→Mūtra, Puriṣa
Dhātu mala→Kapha, Pitta
Agni : *Jaṭharāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Annavaha*
Srotoduṣṭi : *Saṅga & Vimārgagamana, Atipravṛtti*
Adhiṣṭhāna : *Grahaṇī*
Udbhavasthāna : *Grahaṇī*
Vyaktasthāna : *Grahaṇī*
Sancārasthāna : *Mahā Srotas, Annavaha Srotas*
Svabhāva : *Cirakāri*
Prabhāva : *Mahāroga, Sudustura (difficult to treat)*
Roga Mārga : *Abhyantara*
Roga bheda : *Vātaja, Pittaja, Kaphaja & Sannipātaja*

सामान्य लक्षण (Sāmānya Lakṣaṇa)

अतिसृष्टं विबद्धं वा द्रवं तदुपदिश्यते ।
तृष्णारोचकवैरस्यप्रसेकतमकान्वितः ॥

शूनपादकरः सास्थिपर्वरुक् छर्दनं ज्वरः ।
लोहामगन्धिस्तिक्ताम्ल उद्गारश्चास्य जायते ॥

C.S.Ci. 15/53-54

अथ जाते भवेज्जन्तुः शूनपादकरः कृशः ।
पर्वरुग्लौल्यतृद्छर्दिज्वरारोचकदाहवान् ॥
उद्गिरेच्छुक्ततिक्ताम्ललोहधूमामगन्धिकम् ।
प्रसेकमुखवैरस्यतमकारुचिपीडितः ॥

S.S.Utt. 40/174-175

General signs and symptoms of Grahaṇī roga are as follows : Patient passes frequent watery stools or constipated associated with thirst, anorexia, distaste in mouth, excessive salivation, and feeling of darkness. Besides, oedema on feet and hands, pain in bones & joints, vomiting, fever, eructations have metallic or *Āma* or smoke smell; sour, bitter, & vinegar eructations; emaciation, greediness, burning sensation inside and breathlessness.

वातज ग्रहणी रोग हेतु, संप्राप्ति एवं लक्षण (Vātaja Grahaṇī Roga Hetu, Samprāpti Evam Lakṣaṇa)

कटुतिक्तकषायातिरुक्षशीतलभोजनैः ।
प्रमितानशनात्यध्ववेगनिग्रहमैथुनैः ॥
करोति कुपितो मन्दमग्निं संछाद्य मारुतः ।
तस्यान्नं पच्यते दुःखं शुक्तपाकं खराङ्गता ॥
कण्ठास्यशोषः क्षुत्तृष्णा तिमिरं कर्णयोः स्वनः ।
पार्श्वोरुवङ्क्षणग्रीवारुजोऽभीक्ष्णं विसूचिका ॥
हृत्पीडा कार्श्यदौर्बल्यं वैरस्यं परिकर्तिका ।
गृद्धिः सर्वरसानां च मनसः सदनं तथा ॥
जीर्णे जीर्यति चाध्मानं भुक्ते स्वास्थ्यमुपैति च ।
स वातगुल्महृद्रोगप्लीहाशङ्की च मानवः ॥
चिराद्दुःखं द्रवं शुष्कं तन्वामं शब्दफेनवत् ।
पुनः पुनः सृजेद्वर्चः कासश्वासार्दितोऽनिलात् ॥

C.S.Ci. 15/59-64

Vātaja Grahaṇī Roga manifest due to following etiological factors- Excessive consumption of pungent, bitter, astringent, excessively unctuous & cold food substances, less intake of food, fasting too much, travelling on foot, suppression of natural urges and excessive sexual intercourse.

संप्राप्ति (Samprāpti)

Due to etiological factors *Vāta* gets aggravated and surrounds *Agni* as a result *Agni* becomes sluggish and manifest *Vātaja Grahaṇī.*

लक्षण (Lakṣaṇa)

Food digested with difficulty, hyperacidity, roughness in body, dryness of throat & mouth, hunger, thirst, blurred vision, tinnitus, frequent pain in sides of chest, thighs, pelvic region and neck, *Visūcikā,* pain in cardiac region, emaciation, weakness, distaste in mouth, cutting pain in abdomen, desire to take all taste food items, lassitude, flatulence after & during the process of digestion, temporary relief after taking food, patient suspects as if he is suffering from *Vātagulma, Hṛdroga* or *Plīhā roga.* Patient passes stool frequently with difficulty, which is liquid mixed with hard stool, thin, *Āma* associated with sound & froathyness. Patient also suffers from cough & dyspnoea.

पित्तज ग्रहणी रोग हेतु एवं संप्राप्ति (Pittaja Grahaṇī roga hetu evam Samprāpti)

कट्वजीर्णविदाह्यम्लक्षाराद्यैः पित्तमुल्बणम् ।
अग्निमाप्लावयद्धन्ति जलं तप्तमिवानलम् ॥

C.S.Ci. 15/65

Pittaja grahaṇī roga manifest if patient adopts following etiological factors : Intake of pungent, uncooked food; eating during indigestion, food substances which induce burning, sour, alkaline food etc. Due to etiological factors *Pitta* gets aggravated and extinguishes *Agni,* like hot water causes extinction of physical fire.

पित्तज ग्रहणी रोग लक्षण (Pittaja Grahaṇī roga Lakṣaṇa)

सोऽजीर्णं नीलपीताभं पीताभः सार्यते द्रवम् ।
पूत्यम्लोद्गारहृत्कण्ठदाहारुचितृडर्दितः ॥

C.S.Ci. 15/66

Patient passes stool, which is watery, undigested, either bluish-yellow or yellow in colour, foetid and sour eructations, burning sensation in the cardiac region & throat, anorexia & thirst.

कफज ग्रहणी रोग हेतु, संप्राप्ति एवं लक्षण (Kaphaja Grahaṇī Roga Hetu, Samprāpti Evam Lakṣaṇa)

गुर्वतिस्निग्धशीतादिभोजनादतिभोजनात् ।
भुक्तमात्रस्य च स्वप्नाद्धन्त्यग्निं कुपितः कफः ॥
तस्यान्नं पच्यते दुःखं हृल्लासच्छर्द्यरोचकाः ।
आस्योपदेहमाधुर्यकासष्ठीवनपीनसाः ॥
हृदयं मन्यते स्त्यानमुदरं स्तिमितं गुरु ।
दुष्टो मधुर उद्गारः सदनं स्त्रीष्वहर्षणम् ॥
भिन्नामश्लेष्मसंसृष्टगुरुवर्चःप्रवर्तनम् ।
अकृशस्यापि दौर्बल्यमालस्यं च कफात्मके ॥

C.S.Ci. 15/67-70

Kaphaja Grahaṇī Roga manifest by following etiological factors : Excess consumption of food, which is heavy, too unctuous, cold etc; over eating and sleeping immediately after meals.

संप्राप्ति (Samprāpti)

Due to etiological factors *Kapha* gets aggravated and extinguishes *Agni* & manifest *Kaphaja Grahaṇī Roga.*

लक्षण (Lakṣaṇa)

Digestion of food with difficulty, nausea, vomiting, anorexia, sliminess and sweetishness in mouth, cough, spitting sputum, rhinitis, feeling of sluggishness in cardiac region, patient feels his abdomen is still & heavy, sweetish eructations having foul smell, general debility, lack of desire towards women, patient passes stool which is split into pieces, mixed with *Āma* & *Mucus* and heavy; without emaciation patient feels weak & lassitude.

सन्निपातज ग्रहणी रोग हेतु, संप्राप्ति एवं लक्षण (Sannpātaja Grahaṇī Roga hetu, Samprāpti Evam Lakṣaṇa)

पृथग्वातादिनिर्दिष्टहेतुलिङ्गसमागमे ।
त्रिदोषं निर्दिशेत्तेषां भेषजं शृण्वतः परम् ॥

C.S.Ci. 15/72

If person adopts such etiological factors, which is capable of aggravating all the three *Doṣās* simultaneously leading to suppression of *Agni* and manifest signs & symptoms of all the *Doṣās* is known as *Sannipātaja Grahaṇī Roga.*

संग्रहग्रहणी एवं घटीयन्त्र ग्रहणी (Sangraha grahaṇī evam ghaṭi yantra grahaṇī)

अन्त्रकूजनमालस्यं दौर्बल्यं सदनं तथा ।
द्रवं शीतं घनं स्निग्धं सकटीवेदनं शकृत ॥
आमं बहु सपैच्छिल्यं सशब्दं मन्दवेदनम् ।
पक्षान्मासाद्दशाहाद्वा नित्यं वाऽप्यथ मुञ्चति ॥
दिवा प्रकोपो भवति रात्रौ शान्तिं व्रजेच्च या ।
दुर्विज्ञेया दुश्चिकित्स्या चिरकालानुबन्धिना ॥
सा भवेदामवातेन संग्रहग्रहणी मता ।
स्वपतः पार्श्वयोः शूलं गलज्जलघटीध्वनिः ।
तं वदन्ति घटीयन्त्रमसाध्यं ग्रहणीगदम् ॥

M.Ni. 4

Sangrahagrahaṇī is caused by Āma and vāta and it manifest signs and symptoms are as follows : Intestinal gurgling; lassitude; debility; general malaise; passes stool which is liquid, cold, solid, unctuous, associated with pain in low back; patient voids stool with excessive *Āma*, sliminess, sound associated with mild pain. This diseases repeats once in 15 days, 30 days, 10 days or once in a day. Disease aggravates during day time and pacifies in night time. It is very difficult for diagnosis & management. Patient suffers from this entity for longer period of time.

Ghaṭiyantra grahaṇī is characterised by excessive sleeping, pain in sides of chest, and produces the sound like that of water pouring out of pot while defecating and it is incurable.

Prognosis of Grahaṇī roga

दोषं सामं निरामं च विद्यादत्रातिसारवत् ॥
लिङ्गैरसाध्यो ग्रहणीविकारो यैस्तैरतीसारगदो न सिध्येत् ।
वृद्धस्य नूनं ग्रहणीविकारो हत्वा तनुं नैव निवर्तते च ॥
बालके ग्रहणी साध्या यूनि कृच्छ्रा समीरिता ।
वृद्धे त्वसाध्या विज्ञेया मतं धन्वन्तरेरिदम् ॥

M.Ni. 4/18-20

The digested and undigested state of this disease are to be known like *Atisāra.* Prognosis is like that of *Atisāra.* If an old person is afflicted by *Grahanīroga,* it will not leave him even after death. *Grahaṇī roga* in children is curable, it is difficult for management in young and it is incurable in the old, according to *Dhanvantari.*

Differentiate between Grahaṇi Doṣa & Grahaṇīgada/ Grahaṇī Roga

S.No.	Grahaṇi Doṣa	Grahaṇīgada/ Grahaṇī Roga
1	*Grahaṇi Doṣa* refers to group of diseases originated due to vitiation of *Grahaṇī.*	*It is one variety of Grahaṇī Doṣa* There are four varieties of *Grahaṇī roga* namely
2	The word '*Grahaṇī Doṣa*' includes the following diseases *C.S.Ci. 15/71,C.S.Ci. 15/38-41 (Cakrapāṇi)* A) *Grahaṇi roga or Grahaṇī gada* B) *Agnimāndhya* C) *Ajīrṇa* D) *Mandāgni* E) *Tīkṣṇāgni* F) *Viṣamāgni etc.*	*Vātaja, Pittaja, Kaphaja & Sannipātaja* are described

Differential diagnosis of Atisāra and Grahaṇī Roga and Pravāhikā

S.No.	Atisāra	Pravāhikā	Grahaṇī Roga/Grahaṇī Gada
Doṣa	*Vāta & āpadhātus* *Apa dhātus* अपान्धातुःकायद्रवः श्लेष्मपित्तरसादिकः। अन्ये तु कफदुष्टं रसं स्वेदं चापान्धातुशब्देनाहुः। *S.S.Utt. 40/138 Dalhaṇa*	*Vāta & Balāsa (Kapha)* एतेनैतदुक्तं भवति-यत्र रोगे वातप्रेरितं कफं पुरुषः प्रेरयति, तां प्रवाहिकां कफं प्रवाहमाणस्य प्रेर्यमाणस्य तज्ज्ञाः प्रवदन्तीति सम्बन्धः *S.S.Utt. 40/138 Dalhaṇa*	*Vāta* and *agni* plays a vital role
Etiology	*Krimidoṣa* is mentioned as etiology	*Krimidoṣa* is not mentioned under etiology	*Agniduṣṭi* etiological factors mainly responsible for the development of this diasease
Etiology	Psychological factors like *Śoka, Bhaya* as a etiological factors	Such factors are not mentioned	Psychological factors like *Śoka, Bhaya* as a etiological factors
Etiology	Independently develop as a disease due to exposure to etiological factors	Independently develop as a disease due to exposure to etiological factors	It may manifest independently or after *Atisāra* due to consumption of *agni* vitiating etiological factors
Classification	*According to Suśruta Atisāra* is classified into six types i.e. *1. Vātaja, 2. Pittaja, 3. Kaphaja, 4. sannipātaja, 5. Śokaja & 6. Āmaja;* Some people says it is classified into various types but *Kāśirāja* says it is not right, because it has many different stages of the *Doṣās* and appear at various times during the	*1. Vātaja, 2. Pittaja, 3. Kaphaja* and *4. Raktaja Pravākrikā* are described along with their symptomatology & these may be considered as classification of *Pravāhikā.*	There are four varieties of *Grahaṇī roga* namely *Vātaja, Pittaja, Kaphaja & Sannipātaja are mentioned*

	course of the disease. *According to Caraka Atisāra* is classified into five types i.e. *1. Vātaja, 2. Pittaja,* *3. Kaphaja, 4. Sannipātaja &* *5. Āgantuja (Bhayaja & Śokaja).*		
Classification	It is classified briefly into two types i.e. *Sāma* & *Nirāma* and *Sāṣṛk* mixed with blood & *Nirāsṛk* not mixed with blood. *A.H.Ni.8̃/13-14*	Such classification is not mentioned	Such classification is not mentioned
Classification	These six varieties of *Atisāra* are subdivided into thirty more varieties depending on the predominance of one or the other *Doṣa*. If six varieties are combined with them, their numbers becomes thirty six and are manifested along with their complications. *C.S.Si.8̃/21*	Such classification is not mentioned	Such classification is not mentioned
Pūrva rupa	Premonitory signs & symptoms of *Atisāra* are pricking type of pain in heart, umbilicus, rectum, lower abdomen and flanks; general malaise; non elimination of flatus, constipation, distension of abdomen and indigestion.	Premonitory signs & symptoms of *Pravāhikā* are not mentioned	Premonitory signs & symptoms of *Grahaṇī roga* are thirst, laziness, diminution of strength, burning sensation during digestion of food, delayed digestion, heaviness in body, general malaise, exhaustion, anorexia, cough, ringing inside the ears and gurgling noise in the abdomen.
Clinical feature	No oedema on feet and hands, No history of vomiting, fever, eructation's(metallic or *Āma* or smoke smell); no history of sour, bitter, & vinegar eructation's No history of emaciation,	No oedema on feet and hands, No history of vomiting, fever, eructation's(metallic or *Āma* or smoke smell); no history of sour, bitter, & vinegar eructation's No history of emaciation,	Patient passes frequent watery stools or constipated associated with thirst, anorexia, oedema on feet and hands, pain in bones & joints, vomiting, fever, eructations have metallic or *Āma* or smoke smell; sour, bitter, & vinegar eructations; emaciation, greediness, burning sensation inside and breathlessness are observed
Nature	Acute onset	Acute onset	Chronic
Nivṛtti lakṣaṇa	*Atisāra nivṛtti lakṣaṇa* are mentioned *Sannipātaja Atisāra* is very difficult to treat and	*Pravāhikā nivṛtti lakṣaṇa* are mentioned No such descriptions available	*Grahaṇī roga nivṛtti lakṣaṇa* are mentioned *Grahaṇī roga* in children is curable, it is difficult for

	incurable in young & old persons		management in young and it is incurable in the old, according to *Dhanvantari.*
Contemporary	It may be correlated to Diarrhea	Based on the above clinical features it may be correlated to inflammatory bowel disease (IBD) & it is a group of inflammatory conditions of the colon and small intestine, Shigellosis also known as Bacillary Dysentery & Amebiasis	*Grahaṇī roga* may be compared to irritable bowel syndrome & Celiac disease, also known as celiac sprue or gluten-sensitive enteropathy, acid peptic disorders & Gastroesophageal reflux disease (GERD)

Differentiate Doṣaja Grahaṇī Roga

S.No	**Vātaja**	**Pittaja**	**Kaphaja**	**Sannipātaja**
Etiology	Excessive consumption of pungent, bitter, astringent, excessively unctuous & cold food substances, less intake of food, fasting too much, travelling on foot, suppression of natural urges and excessive sexual intercourse.	Intake of pungent, uncooked food; eating during indigestion, food substances which induce burning, sour, alkaline food etc. Due to etiological factors *Pitta* gets aggravated and extinguishes *Agni,* like hot water causes extinction of physical fire.	Excess consumption of food, which is heavy, too unctuous, cold etc; over eating and sleeping immediately after meals.	If person adopts such etiological factors, which is capable of aggravating all the three *Doṣās* simultaneously leading to suppression of *Agni* and manifest *Sannipātaja*
Clinical feature	Patient passes stool frequently with difficulty, which is liquid mixed with hard stool, thin, *Āma* associated with sound & froathyness	Patient passes stool, which is watery, undigested, either bluish-yellow or yellow in colour, foetid and sour eructations, burning sensation in the cardiac region & throat, anorexia & thirst	patient passes stool which is split into pieces, mixed with *Āma* & *Mucus* and heavy	patient passes stool which is mixture of features of all the three *doṣās*
Nature of pain	Pain in cardiac region	Burning sensation in the cardiac region & throat	Feeling of sluggishness in cardiac region,	Pain, burning and feeling of sluggishness in cardiac region
Eructations	Hyperacidity and Acid reflux	Foetid and sour eructations	Sweetish eructation's having foul smell	Acid reflux, foetid and sour eructation& sweetish eructation's having foul smell
Contemporary	Based on above clinical features *Vātaja Grahaṇī* may be correlated with Celiac disease/celiac	Based on above clinical features *Pittaja Grahaṇī* may be correlated with acid	Based on the above clinical features *Kaphaja Grahaṇī* may be correlated with	Based on above clinical features *Sannipātaja Grahaṇīroga* may be

	sprue or guten sensitive enteropathy, tropical sprue, Irritable Bowel Syndrome (IBS), PEM (protien energy malnutrition) etc.	peptic disorders.	Gastroesophageal reflux disease (GERD), Malabsorption syndrome.	correlated with IBS-M (Mixed diarrhoea and constipation), IBS- U (unclassified).

उपशय/पथ्य (Upaśaya/Pathya)

Auṣadha- *Daśamūlādya ghṛta, Tryūṣaṇādya ghṛta, Pañcamūlādya ghṛta* and *Cūrṇa, Citrakādya guṭikā, Pippalādya Cūrṇa, Maricyādi Cūrṇa, Takrāriṣṭa, Tiktaka ghṛta, Nāgarādya Cūrṇa, Bhūnimbādya Cūrṇa, Kirātādya Cūrṇa, Madhūkāsava, Durālabhāsava, Mūlāsava, Piṇḍāsava, Madhvāriṣṭa, Pippalyādi Cūrṇa, Kṣāra Ghṛta, Bhallātakādya Kṣāra, Durālabhādya Kṣāra, Bhunimbādikṣāra, Haridrādya Kṣāra, Kṣāra guṭikā, Tālisapatrādi Cūrṇa, Pañcamūlādi Ghṛta.*

Anna- C.S.Ci. 15/115-117

Soup of *Pañcakola* or the soup of *Mūlaka* prepared by adding *Marica* and ghee or oil, or spiced meat soup of wild animals added with ghee and sour pomegranate & butter, meat soup of carnivorous animals mixed with appetisers are recommended as diet. Buttermilk, sour gruel, wine & *Ariṣṭa* should be taken as post meal drink.

Use of buttermilk (butter milk) with salt is very effective in checking psilosis. The buttermilk is very useful in adjusting the three orders of body including *Vāta*, *Pitta* and *Kapha*. It is light and thus helpful in promoting digestion. It is *Madhura* in *Vipāka* and thus it does not vitiate the *Pitta Doṣa*. It is sour, astringent, hot by nature and thus it controls the *Kapha* contents as well. The fresh buttermilk is smooth and it does not produce burning sensation as well. (B.R. 8/3-4)

The following measures are considered to be beneficial for patients of *Grahaṇī*

Sleeping, vomiting, *Laṅghana* (fasting), taking in meals old *Śāli* rice, old *Ṣaṣṭhi* rice, liquid obtained from the cooked rice, pulse soup of *Masūra* (lentil), *Ādhya* (pigeon peas) and *Mudga* (kidney beans) pulses, curd of cow's milk containing no butter, butter obtained from goat's milk or curd, *Ghṛtam* of goat's milk, cow's milk, curd, sesame oil, liquor, honey, *Kamalakanda, Maulśrī*, both types of pomegranate, fresh fruits of *Kaseru*, fruit and flowers of banana, pulp of fresh *Bilva* fruits, *Śṛīṅgāṭaka, Cāngerī, Vijayā, Kapittha*, bark of *Kuṭaja*, cumin seeds, buttermilk, leaves of *Kancṭa, Niśānna, Jātīphala, Jambū*, coriander, *Tinduka, Mahānimba, Mañjiṣṭhā, Pelava*, meat of carnivorous birds, meat soup of rabbit, *Eṇa* and *Tittira* (partridge) and all types of small fish, *Khuddiśa* (one type of fish which flies for a short period), *Madhuralikā* (type of fish), *Khalīś* (type of fish), all types of sour liquids. One can also heat up the patient around the areas two fingers up and two fingers down of belly and also at the roots of backbone. It also helps the patient of *Grahaṇī*. (B.R. 8/622-625)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following are considered to be harmful in cases of *Grahaṇī*: blood letting, roaming during night, drinking excessive water, bathing, intercourse, stopping flow of excretions, nasal therapy, application of collyarium, *Svedana* therapy, smoking, heavy work, uncomptiable food, sun bathing or enjoying fire heat, taking food items prepared from wheat, white beans, peas, *Urada* (pulse soup), barley, ginger, *Catraka* (mushroom), *Rājamāṣa, Upodikā, Vāstuka, Kākamācī*, ash gourd or *Kāśīphala, Tumbī*, ripe drum stick, potato, *Arui*, and other tubers, betel leaves, sugarcane, berry, mango, snake cucumber (*Karkaṭī*), betel nut, garlic, *Kānji, Sauvira* (barley and wheat's *Kānji*), *Tuṣodaka* (*Kānji* of *yava* along with its peels), milk, jaggery, buttermilk, coconut fruit or water, *Punarnavā*, fruit of *Kaṅṭakārī*, bamboo shoots, all types of leafy cuisine, contaminated water, cow's urine, *Kastūrī, Yavakṣāra*, all types of laxative liquids, *Drākṣā* and like sour eatables, salt, taking heavy to digest meals, all types of pudding and *Puri*. (B.R. 8/617-621)

MCQs

1. *Bhuktamamam eva vimunchati, muhurbadhammuhurdravam* is the feature of
   - a. *Grahani*
   - b. *Atisara*
   - c. *Chardhi*
   - d. *Pravahika*
2. In *Samgrahagrahani, malapravrthi* occurs in
   - a. *Paksha, Masa,* 10 days or daily
   - b. 6 months
   - c. 8 months
   - d. Years
3. *Diva prakopobhavatiratrousanti* is the feature of
   - a. *Vatajagrahani*
   - b. *Tridoshagrahani*
   - c. *Samgrahagrahani*
   - d. *Ghatiyantragrahani*
4. Prognosis of *Ghatiyantragrahani*
   - a. *Dussaha*
   - b. *Asadya*
   - c. *Yapya*
   - d. *Krichrasadya*
5. *Parswasoola* while sleeping is the feature of
   - a. *Samgrahagrahani*
   - b. *Ghatiyantragrahani*
   - c. *Vatajagrahani*
   - d. *Tridoshagrahani*
6. *Kara padaswayathu* is see in
   - a. *Grahanipurvaroopa*
   - b. *Grahaniupadrava*
   - c. *Kaphajagrahani*
   - d. *Grahaniroopa*
7. *Streeshuaharshanam* is the feature of
   - a. *Kaphajaatisara*
   - b. *Vatajaatisara*
   - c. *Vatajagrahani*
   - d. *Kaphajagrahani*
8. *Akrisayaapidourbalyam* is the feature of
   - a. *Kaphaja grahani*
   - b. *Kaphaja atisara*
   - c. *Shokaja atisara*
   - d. *Vataja grahani*
9. *Jeernejeeryati cha aadhmanambhukteswasthyam* is the feature of
   - a. *Vatajagrahani*
   - b. *Parinamasula*
   - c. *Annadravasula*
   - d. *Teekshnagni*
10. Types of *Grahanigada*
    - a. 3
    - b. 4
    - c. 5
    - d. 6
11. *Vatahridrogagulmaarsapleehapandutvasankina* is the feature of
    - a. *Vata Grahani*
    - b. *Arsas*
    - c. Both a & b
    - d. *Udävarta*

Answer

1.	a	2.	a	3.	c
4.	b	5.	b	6.	d
7.	d	8.	a	9.	a
10.	b	11.	a		

CHAPTER
7

Visuchika, Alasaka and Vilambhika

Definition of visūcikā

It is caused by stretching of walls of hollow viscera, innervated by unmyelinated fibres. It is diffuse and intermittent, dull aching and colicky in nature. Patients keep tossing on the bed. It is felt in the abdominal region which correlates to the somatic segment of embryonic region. Foregut, midgut and hindgut structures relate to upper, middle and lower abdomen, respectively. Visceral pain can be perceived away from actual disease process, i.e. pain of acute appendicitis is felt around umbilicus initially as it corresponds to T10 somatic distribution.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

विविधसूचीतुल्यत्वाद्विसूचिका । *A.H.Su. 8/7 (Hemādri)*

One who experiences different types of pain like pricking, colicky etc is known as *Visūcikā.*

विविधानां विकारणां सूचिका-विसूचिकेत्युच्यते ॥
A.H.Su. 8/7 (Aruṇdatta)

One who experiences varied types of pricking, cramp pain is known as *Visūcikā.*

यस्माद्गात्राणि सूचीभिरिव विध्यति, तस्माद्विसूचिकेत्युच्यते ।
A.H.Su. 8/7 (Aruṇdatta)

Development of pain all over the body like being pricked by needles is known as *Visūcikā.*

विशेषण सूचीभिरिव मर्माणि पीडयतीति विसूची ॥
Āḍd.Śa.Prā. 7/11

Manifestation of pricking pain in the vital body parts is known as *Visūcikā.*

बाहुल्याद्वायुः सूचीभिरिव तुदन् इति विसूचीनिरुक्तिः ॥
M.Ni. 6/16 (Madhu)

Pricking type of pain due to aggravation of *Vāta* is known as *Vīsūcikā.*

विविधैर्वेदनोद्भेदैर्वाय्वादिभृशकोपतः ।
सूचीभिरिव गात्राणि विध्यतीति विसूचिका ॥
A.H.Su. 8/7-8

Aggravation of *Vātadi doṣas* causing different types of pain as though being pricked by needles; therefore it is called as *Visūcikā.*

सूचीभिरिव गात्राणि तुदन् सन्तिष्ठतेऽनिलः ।
यस्याजीर्णेन सा वैद्यैरुच्यतेति विसूचिका ॥
S.S.Utt. 56/4

Suffering from pricking pain in the body due to the vitiation of *Vāta* due to indigestion is called *Visūcīka* by the physicians.

Definition of Alasaka

It results from irritation of parietal peritoneum caused by inflammation, infection or chemical reaction. It is supplied by myelinated nerve fibres. It is localised and constant. As the disease process evolves and irritates parietal peritoneum, we can elicit tenderness, guarding and rigidity. The patient prefers to lie immobile.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

It is localised and constant. As the disease process evolves and irritates parietal peritoneum, we can elicit tenderness, guarding and rigidity. The patient prefers to lie immobile.

अलस्त्वं-निष्क्रियत्वम् । *A.H.Su. 86 (Hemādri)*

The term *Alasa* means *Niṣkriyatva* (inactive).

दोषस्थिरत्वनिमित्तादलसक संज्ञा ॥ *Āḍd.Śa.Prā. 7/10*
अलसक इति दोषस्थिरत्वनिमित्ता सञ्ज्ञा ॥
Ma.Ni. 6/19 (Madhu)

Immobile state of abdomen is called as *Alasaka.*

कोष्ठेस्थितोऽलसीभूतस्तेन चालसकः समृतः ॥
Āḍd.Śa.Prā. 7/10

That which causes ingested food to remain idle in the *Koṣṭha* (stomach) is called *Alasaka.*

प्रयाति नोर्ध्वं नाधस्तादाहरो न च पच्यते ।
आमशयेऽलसीभूतस्तेन सोऽलसकः स्मृतः ॥
A.H.Su. 86/Ma.Ni. 6/19-20 (Madhu)

The condition where the ingested food to remain idle in the stomach without undergoing digestion and either moves upwards (vomiting) nor downwards (purgation) is known as *Alasaka*.

Definition of Vilambikā

This is a kind of indigestion. It occurs due to profound accumulation of *āma* inside the channels. The disease is produced jointly by vitiated *kapha* and *vāta* and has all the symptoms of *āma*. Hence, its treatment is similar to that of *āma*. (*AH*. *Sū*. 8. 28).

The place where *āma* gets lodged produce not only pain but also becomes the site for occurence of several diseases. This situation develops as a result of the action of *doṣa* that travel across the body associated with *āma*. (*SS*. *Ut*. 56. 9).

A type of intestinal disorder characterized by stagnation of food ingested. If the ingested food gets vitiated by the aggravated *kapha* and *vāyu*, it moves neither upwards nor downwards. This condition, according to ancient scholars of medicine, is called *vilambikā*. It is extremely difficult to cure. The ingested food, when vitiated by *kapha* and *vāta* does not move upwards or downwards. Such symptoms has been identified as the *Vilambikā* which is very tough to manage according to learned ones.

अजीर्णमामं विष्टब्धं विदग्धञ्च यदीरितम् ।
विसूच्यलसकौ तस्माद्भवेच्चापि विलम्बिका ॥
S.S.Utt.56/3

Visūcikā, Alasaka & Vilambikā manifest due to afflictions of *Āmājīrṇa, Viṣṭabdhājīrṇa & Vidagdhājīrṇa.*

विसूचिका (Visūcikā)

सूचीभिरिव गात्राणि तुदन् सन्तिष्ठतेऽनिलः ।
यस्याजीर्णेन सा वैद्यैरुच्यतेति विसूचिका ॥
न तां परिमिताहारा लभन्ते विदितागमाः ।
मूढास्तामजितात्मानो लभन्ते कलुषाशयाः ॥
S.S.Utt. 56/4-5

तं द्विविधमामप्रदोपमाचक्षते भिषजः-विसूचिकाम्, अलसकं च। तत्र विसूचिकामूर्ध्वं चाधश्च प्रवृत्तामदोषां यथोक्तरूपां विद्यात् ॥
C.S.Vi. 2/10-11

Excessively aggravated *Vāta* consequent to *Ajīrṇa* gives rise to pricking pain in the body is known as *Visūcikā*. One who consumes proper amount of wholesome food and adhering to dietetic rules & regulations will not suffer from *Visūcikā*. It manifest in those individuals who have no control over themselves and indulges in such activities which vitiates the stomach as a result *Visūcikā* manifest.

विसूचिका लक्षण (Visūcikā Lakṣaṇa)

It is characterised by fainting, diarrhoea, vomiting, thirst, abdominal pain, giddiness, rigidity of the extremities, yawning, burning sensation, abnormal complexion, tremors, pain in cardiac region, & headache. *Āmadoṣa* manifest two pathological conditions i.e. *Visūcikā* and *Alasaka*. *Visūcikā* is characterised by expulsion of undigested food from upper channels and lower channels.

अलसक (Alasaka)

कुक्षिरानह्यतेऽत्यर्थं प्रताम्यति विकूजति ।
निरुद्धो मारुतश्चापि कुक्षौ विपरिधावति ॥
वातवर्चोनिरोधश्च कुक्षौ यस्य भृशम्भवेत् ।
तस्यालसकमाचष्टे तृष्णोद्गारावरोधकौ ॥
S.S.Utt. 56/7-8

अलसकमुपदेक्ष्यामदुर्बलस्याल्पाग्नेर्बहुश्लेष्मणो वातमूत्रपुरीषवेगविधारिणः स्थिरगुरुबहुरूक्षशीतशुष्कान्नसेविनस्तदन्नपानमनिलप्रपीडितं श्लेष्मणा च विबद्धमार्गमतिमात्रप्रलीनमलसत्वान्न बर्हिमुखीभवति, ततश्छर्द्यतीसारवर्ज्यान्यामप्रदोषलिङ्गान्यभिदर्शयत्यतिमात्राणि। *C.S.Vi. 2/12*

Severe distension of the abdomen, the patient becomes unconscious and moons, *Vāta* getting obstructed moves upwards in the stomach, there is obstruction to flatus, faeces & accumalation in more quantity inside the abdomen, accumpanined with thirst and excessive belchings is known as *Alasaka*.

If debilitated individuals suffering from *Mandāgni* and excessive *Kapha,* suppresses the urge for passing flatus, urine & stool, and consumes compact, heavy, ununctous, cold & dried food in excess; get pressed by *Vāta* and their passage being obstructed by *Kapha*. Because of the sluggishness caused by these factors it is not possible for the undigested food product to come out; then they exhibit excessively the symptom of over affliction of *Āma* except vomiting

दण्डालसक (Daṇḍālasaka)

अतिमात्रप्रदुष्टाश्च दोषाः प्रदुष्टामबद्धमार्गास्तिर्यग्गच्छन्तः कदाचिदेव केवलमस्य शरीरं दण्डवत् स्तम्भ्यन्ति, ततस्तं दण्डालसकमसाध्यं ब्रुवते । *C.S.Vi. 2/12*

Excessively aggravated and vitiated *Doṣās* moves obliquely, when their passage is blocked by over vitiated *Āma* and make the body of the patient rigid like a stiff is known as *Daṇḍālasaka.*

आमविष (Āmaviṣa)

Described in first volume of my book under *Āma* chapter.

विलम्बिका (Vilambikā)

दुष्टन्तु भुक्तं कफमारुताभ्यां प्रवर्त्तते नोद्ध्वमधश्च यस्य ।
विलम्बिकां तां भृशदुश्चिकित्स्यामाचक्षते शास्त्रविदः पुराणाः ॥
S.S.Utt. 56/9

Food getting vitiated by *Kapha* & *Vāta,* does not come out either from downward or from upward channels and waste products remain for longer period of time in the *Mahāsrotas* is known as *Vilambikā* and which is difficult to treat.

Signs and symptoms after consumption of in improper quantity of food

अमात्रावत्त्वं पुनर्द्विविधमाचक्षतेहीनम्, अधिकं च । तत्र हीनमात्रमाहारराशिं बलवर्णोपचयक्षयकरमतृप्तिकरमुदावर्तकरमनायुष्यवृष्यमनौजस्यं शरीरमनोबुद्धीन्द्रियोपघातकरं सारविधमनम–लक्ष्म्यावहमशीतेश्च वातविकाराणामायतनपाचक्षते, अतिमात्रं पुनः सर्वदोषप्रकोपणमिच्छन्ति कुशलाः । यो हि मूर्तानामाहारजातानां सौहित्यं गत्वा द्रवैस्तृप्तिमापद्यते भूयस्तस्यामाशयगता वातपित्तश्लेष्माणोऽभ्यवहारेणातिमात्रेणातिप्रपीड्यमानाः सर्वे युगपत् प्रकोपमापद्यन्ते, ते प्रकुपितास्तमेवाहारराशिमपरिणतमाविश्य कुक्ष्येकदेशमन्नार्श्रिता विष्टम्भयन्तः सहसा वाऽप्युत्तराधराभ्यां मार्गाभ्यां प्रच्यावयन्तः पृथक् पृथागिमान् विकारानभिनिर्वर्तयन्त्यतिमात्रभोक्तुः तत्र वातः शूलानाहाङ्गमर्दमुखशोष मूर्च्छाभ्रमाग्निवैषम्यापार्श्वपृष्ठकटिग्रहसिराकुञ्चनस्तम्भनानि करोति, पित्तं पुनर्ज्वातीसारान्तर्दाह तृष्णामदभ्रमप्रलपनानि, श्लेष्मा तु छर्द्यरोचकाविपाकशीतज्वरालस्यगात्रगौरवाणि ॥ *C.S.Vi. 2/7*

Improper quantity of food is subdivided into two types namely deficient and excess.

Deficient quantity of food produces following signs & symptoms are as follows : Loss of strength, complexion & development; emaciation; unsaturation; upward movement of *Vāta;* diminished longevity, virility and ojas (immunity); damage to body, mind, intellect and senses; impairment of *Sāra;* development of inauspicious conditions and causative factor for eighty types of *Vātaja Vikara.*

Effect of excess quantity of food

Excessively consumed food aggravates all the three *Doṣās.* One who consumes solid food in sufficient quantity and then takes liquid food in excessive quantity, all the three *Doṣās* get too much compressed and simultaneously aggravated. These excacerbated *Doṣās* enters into the undigested food situated in a portion of belly and cause distension of abdomen or instantaneously moves through upward & down tracts separately to produce following signs and symptoms-

Vāta produces colic pain; constipation; malaise; dryness of mouth; fainting; giddiness; irregular digestion; stiffness in sides of chest, back and waist; constriction & hardening of blood vessels. *Pitta* produces fever, diarrhoea, internal burning sensation, thirst, intoxication, giddiness & delirium. *Kapha* produces vomiting, anorexia, indigestion, fever with cold, lassitude & heaviness of body.

MCQs

1. *Dosha* predominance of *Vilambhika*
   - a. *Vatika* b. *Vatakaphaja*
   - c. *Vatapittaja* d. *Tridoshaja*
2. Prognosis of *Vilambhika*
   - a. *Asadya*
   - b. *Kashtasadya*
   - c. *Brisaduschikitsya*
   - d. *Yapya*
3. *Vishuchika upadrava*

a. *Nidranasa, Arati & Mutraghata*
b. *Nidra, Glani, ati pravritti* of *mutra*
c. *Tama, tandra & glani*
d. *Murchha, tandra, glani*

4. *Kukshiranahyatheatyartham* is the feature of
a. *Alasaka* b. *Amaajeerna*
c. *Vilambika* d. *Gulma*

5. Number of *Vishuchika upadrava* as per *Madavanidana*
a. 4 b. 5
c. 6 d. 7

6. Which among the following is not the *Asadyalakshana* of *Vishuchika & Alasaka*
a. *Syavadanta &oshta*
b. *Abhyantaraayatanetra*
c. *Kshamaswara*
d. *Vishamajwara*

7. Suchibhirivagatranitudyan is the feature of
a. *Vilambika* b. *Gulma*
c. *Vishuchika* d. *Sula*

8. Which among the following is not the *lakshana* of *Alasaka*
a. *Vatavarchonirodha*
b. *Trishna*
c. *Udgara*
d. *Na pravarthateurdhwamadhascha*

9. *Kampa* and *hridayaruja* seen in
a. *Vatajaajeerna* b. *Vishuchika*
c. *Anaha* d. *Vilambhika*

Answer

1.	b	2.	c	3.	a
4.	a	5.	b	6.	d
7.	c	8.	d	9.	b

CHAPTER
8

Common GIT Diseases

Ulcerative Dyspepsia

Ulcerative colitis (UC) is an inflammatory bowel disease. It causes irritation, inflammation, and ulcers in the lining of large intestine. The overall estimated IBD population in India in 2010 came out to be 1.4 million, which was the second highest number after the USA (with 1.64 million). Therefore, although the disease prevalence in India is lower than in the West, with a population of more than 120 million, the total IBD population in India is among the largest across the globe.

The primary symptoms of active disease are abdominal pain and diarrhea mixed with blood.Weight loss, fever, and anemia may also occur. Often, symptoms come on slowly and can range from mild to severe. Symptoms typically occur intermittently with periods of no symptoms between flares. Complications may include megacolon, inflammation of the eye, joints, or liver, and colon cancer.

The cause of UC is unknown.Theories involve immune system dysfunction, genetics, changes in the normal gut bacteria, and environmental factors. Rates tend to be higher in the developed world with some proposing this to be the result of less exposure to intestinal infections, or to a Western diet and lifestyle.The removal of the appendix at an early age may be protective. Diagnosis is typically by colonoscopy with tissue biopsies.It is a kind of inflammatory bowel disease (IBD) along with Crohn's disease and microscopic colitis. Dietary changes, such as maintaining a high-calorie diet or lactose-free diet, may improve symptoms.Several medications are used to treat symptoms and bring about and maintain remission, including aminosalicylates as mesalazine or sulfasalazine, steroids, immunosuppressants such as azathioprine, and biologic therapy. Removal of the colon by surgery may be necessary if the disease is severe, does not respond to treatment, or if complications such as colon cancer develop. Removal of the colon and rectum can cure the disease.

Causes of UC

No direct causes for UC are known, but many possible factors such as genetics and stress play a role.

Genetic factors

A genetic component to the cause of UC can be hypothesized based on:

- Aggregation of ulcerative colitis in families
- Identical twin concordance rate of 10% and dizygotic twin concordance rate of 3%
- Ethnic differences in the number of cases
- Genetic markers and linkages

Twelve regions of the genome may be linked to UC, including, in the order of their discovery, chromosomes 16, 12, 6, 14, 5, 19, 1, and 3,] but none of these loci has been consistently shown to be at fault, suggesting that the disorder is influenced by multiple genes. For example, chromosome band 1p36 is one such region thought to be linked to inflammatory bowel disease.

Some of the putative regions encode transporter proteins such as OCTN1 and OCTN2. Other potential regions involve cell scaffolding proteins such as the MAGUK family. Human leukocyte antigen associations may even be at work. In fact, this linkage on chromosome 6 may be the most convincing and consistent of the genetic candidates.

Environmental factors

It is hypothesized that environmental factors contributing to the pathogenesis of ulcerative colitis. These are as below-

Diet: As the colon is exposed to many dietary substances which may affected with inflammation. Dietary factors have been hypothesized to play a role in the pathogenesis of both ulcerative colitis and Crohn's disease. Few studies have investigated such an association of dietary intake and genesis of IBS, which are as below.

1. One study showed no association of refined sugar on the number of people affected of ulcerative colitis.
2. High intake of unsaturated fat and vitamin B6 may enhance the risk of developing ulcerative colitis.
3. Other identified dietary factors that may influence the development and/or relapse of the disease include meat protein and alcoholic beverages.
4. Sulfur has been investigated as being involved in the cause of ulcerative colitis, but this is controversial.Sulfur restricted diets have been investigated in people with UC and animal models of the disease. The theory of sulfur as an etiological factor is related to the gut microbiota and mucosal sulfide detoxification in addition to the diet.

Breastfeeding: Some reports of the protection of breastfeeding in the development of IBD contradict each other. One Italian study showed a potential protective effect.

One study of isotretinoin found a small increase in the rate of UC.

UC as autoimmune disease

Ulcerative colitis is an autoimmune disease characterized by T-cells infiltrating the colon.In contrast to Crohn's disease, which can affect areas of the gastrointestinal tract outside of the colon, ulcerative colitis usually involves the rectum and is confined to the colon, with occasional involvement of the ileum. This so-called "backwash ileitis" can occur in 10-20% of people with pancolitis and is believed to be of little clinical significance. Ulcerative colitis can also be associated with comorbidities that produce symptoms in many areas of the body outside the digestive system. Surgical removal of the large intestine often cures the disease.

Alternative theories

Risk factors

	Crohn's disease	Ulcerative colitis
Smoking	Higher risk for smokers	Lower risk for smokers
Age	Usual onset between 15 and 30 years	Peak incidence between 15 and 25 years

Levels of sulfate-reducing bacteria tend to be higher in persons with ulcerative colitis, which could indicate higher levels of hydrogen sulfide in the intestine. An alternative theory suggests that the symptoms of the disease may be caused by toxic effects of the hydrogen sulfide on the cells lining the intestine.

Infection by mycobacterium avium, subspecies para-tuberculosis, has been proposed as the ultimate cause of both ulcerative colitis and Crohn's disease.

Types of Ulcerative Colitis

The type of ulcerative colitis you have depends on where it's located in your body:

- **Ulcerative proctitis :** It is usually the mildest form. It's only in the rectum, the part of colon closest to anus. Rectal bleeding may be the only sign of the disease.
- **Proctosigmoiditis:** Rectum and the lower end of colon are involved. In this condition patients have bloody diarrhea, belly cramps, and pain. Patients have the urge to poop but would not be able to.
- **Left-sided:** It causes cramps on that side of belly. Patients will also have bloody diarrhea, and might lose weight without trying. Patients will have inflammation from rectum up through the left side of colon.
- **Pancolitis:** It often affects entire colon. It can cause severe bouts of bloody diarrhea, belly cramps, pain, fatigue, and major weight loss.
- **Acute severe ulcerative colitis:** It is rare form of UC. It affects entire colon and causes severe pain, heavy diarrhea, bleeding and fever.

Pathophysiology

	Crohn's disease	Ulcerative colitis
Cytokine response	Associated with Th17	Vaguely associated with Th2

An increased amount of colonic sulfate-reducing bacteria has been observed in some people with ulcerative colitis, resulting in higher concentrations of the toxic gas hydrogen sulfide. Human colonic mucosa is maintained by the colonic epithelial barrier and immune cells in the lamina propria (see intestinal mucosal barrier). N-butyrate, a short-chain fatty acid, gets oxidized through the beta oxidation pathway into carbon dioxide and ketone bodies. It has been shown that N-butyrate helps supply nutrients to this epithelial barrier. Studies have proposed that hydrogen sulfide plays a role in impairing this beta-oxidation pathway by interrupting the short chain acetyl-CoA dehydrogenase, an enzyme within the pathway. Furthermore, it has been suggested that the protective benefit of smoking in ulcerative colitis is due to the hydrogen cyanide from cigarette smoke reacting with hydrogen sulfide to produce the non-toxic isothiocyanate, thereby inhibiting sulfides from interrupting the pathway. An unrelated study suggested that the sulfur contained in red meats and alcohol may lead to an increased risk of relapse for people in remission.

Patients with ulcerative colitis typically present with rectal bleeding, diarrhea, tenesmus (urgent desire to evacuate the bowels but with the passage of little stool), and lower abdominal pain. The severity of disease at clinical presentation is important in determining the appropriate therapy. Peoples with mildly active disease will have fewer than 4 bowel movements daily and no signs of toxicity. Individuals with moderate-severity UC have more frequent bowel movements with bleeding. Approximately 70% of people with ulcerative colitis will have moderately active disease at presentation. People with severely active disease will have signs of toxicity with fever, tachycardia, and anemia. People with fulminant or toxic colitis or toxic megacolon often have more than 10 bowel movements in a day, continuous bleeding, abdominal distention and tenderness, and radiologic evidence of edema and, in some cases, bowel dilation. These people most often require immediate colectomy because 10% have perforated colon at the time of surgery,

Signs and symptoms

Items	Crohn's disease	Ulcerative colitis
Defecation	Often porridge-like, sometimes steatorrhea	Often mucus-like and with blood
Tenesmus	Less common	More common
Fever	Common	Indicates severe disease
Fistulae	Common	Seldom
Weight loss	Often	More seldom

Gastrointestinal

The clinical presentation of ulcerative colitis depends on the extent of the disease process. Patients with ulcerative colitis usually present with diarrhea mixed with blood and mucus, of gradual onset that persists for an extended period (weeks). They may also have weight loss and blood on rectal examination. The inflammation caused by the disease along with the chronic bleeding from the GI tract leads to increased rates of anemia. The disease may be accompanied by different degrees of abdominal pain, from mild discomfort to painful bowel movements or painful abdominal cramping with bowel movements.

Ulcerative colitis is associated with a general inflammatory process that can affect many parts of the body. Sometimes, these associated extra-intestinal symptoms are the initial signs of the disease, such as painful arthritic knees in teenagers, which also may be seen in adults. A diagnosis of UC may not occur until the onset of intestinal manifestations.

Extent of intestinal involvement

Ulcerative colitis is normally continuous from the rectum up the colon. The disease is classified by the extent of involvement, depending on how far the disease extends:

- Distal colitis, potentially treatable with enemas:

- o Proctitis: Involvement limited to the rectum
- o Proctosigmoiditis: Involvement of the rectosigmoid colon, the portion of the colon adjacent to the rectum
- o Left-sided colitis: Involvement of the descending colon, which runs along the person's left side, up to the splenic flexure and the beginning of the transverse colon
- Extensive colitis: Inflammation extending beyond the reach of enemas:
- o *Pancolitis*: Involvement of the entire colon, extending from the rectum to the cecum, beyond which the small intestine begins

In addition to the extent of involvement, people may also be characterized by the severity of their disease.

- Mild disease correlates with fewer than four stools daily, with or without blood, no systemic signs of toxicity, and a normal erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP). Mild abdominal pain or cramping may occur. People with UC may believe they are constipated when in fact they are experiencing tenesmus, which is a constant feeling of the need to empty the bowel accompanied by involuntary straining efforts, pain, and cramping with little or no fecal output.
- Moderate disease correlates with more than four stools daily, but with minimal signs of toxicity. People may display anemia (not requiring transfusions), moderate abdominal pain, and low-grade fever, 38 to 39 ãC (100 to 102 ã̄F).
- Severe disease correlates with more than six bloody stools a day or observable massive and significant bloody bowel movement, and evidence of toxicity as demonstrated by fever, tachycardia, anemia or an elevated ESR or CRP.

Fulminant disease correlates with more than 10 bowel movements daily, continuous bleeding, toxicity, abdominal tenderness and distension, blood transfusion requirement, and colonic dilation (expansion). People in this category may have inflammation extending beyond just the mucosal layer, causing impaired colonic motility and leading to toxic megacolon. If the serous membrane is involved, a colonic perforation may ensue. Unless treated, the fulminant disease will soon lead to death.

Ulcerative colitis - Disease severity

Classification	Description	Prevalence
Mild	•<4 stools/day +/- blood •Normal ESR •No signs for systemic toxicity	22%28%
Moderate	•≥4 stools/day •Minimal signs of toxicity	43%-46%
Severe	•>6 blood stools/day •Fever, tachycardia, anemia, or / ESR	28%-31%
Fulminant	•>10 stools/day, continuous bleeding •Toxicity, abdominal tenderness/distension, transfusion requirement, colonic dilation on x-ray	-9%

ESR = erythrocyte sedimentation rate.

Extra-intestinal manifestation

As UC is believed to have a systemic (i.e., autoimmune) origin, people with UC may present with comorbidities leading to symptoms and complications outside the colon. The frequency of such extra-intestinal manifestations has been reported as between 6 and 47% and it includes:

- Aphthous ulcer of the mouth
- Ophthalmic
- o Iritis or uveitis, which is inflammation of the eye's iris
- o Episcleritis
- Musculoskeletal:
- o Seronegative arthritis, which can be a large-joint oligoarthritis (affecting one or two joints), or may affect many small joints of the hands and feet
- o Ankylosing spondylitis, arthritis of the spine
- o Sacroiliitis, arthritis of the lower spine
- Cutaneous (related to the skin):

- o Erythema nodosum, which is a panniculitis, or inflammation of subcutaneous tissue involving the lower extremities
- o Pyoderma gangrenosum, which is a painful ulcerating lesion involving the skin
- Deep venous thrombosis and pulmonary embolism
- Autoimmune hemolytic anemia
- Clubbing, a deformity of the ends of the fingers
- Primary sclerosing cholangitis, a distinct disease that causes inflammation of the bile ducts

Irritable bowel syndrome

Irritable bowel syndrome (IBS) is a syndromic presentation of group of symptoms-including abdominal pain and changes in the pattern of bowel movements without any evidence of underlying cause. These symptoms occur over a long time, often years.It has been classified into four main types depending on whether diarrhea is common, constipation is common, both are common, or neither occurs very often (IBS-D, IBS-C, IBS-M, or IBS-U respectively). IBS negatively affects quality of life and may result in missed school or work.Disorders such as anxiety, major depression, and chronic fatigue syndrome are common among people with IBS.

In conventional system of medicine the causes of IBS are not clear.Theories include combinations of gut-brain axis problems, gut motility disorders, pain sensitivity, infections including small intestinal bacterial overgrowth, neurotransmitters, genetic factors, and food sensitivity. Onset may be triggered by an intestinal infection, or stressful life event. IBS is a functional gastrointestinal disorder.Diagnosis is based on symptoms in the absence of worrisome features and once other potential conditions have been ruled out.Worrisome features include onset at greater than 50 years of age, weight loss, blood in the stool, or a family history of inflammatory bowel disease. Other conditions that may present similarly include celiac disease, microscopic colitis, inflammatory bowel disease, bile acid malabsorption, and colon cancer.

There is no known cure for IBS in modern medicine.Treatment is carried out to improve symptoms. This may include dietary changes, medication, probiotics, and counseling. Dietary measures include increasing soluble fiber intake, a gluten-free diet, or a short-term diet low in fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs).The medication loperamide may be used to help with diarrhea while laxatives may be used to help with constipation. Antidepressants may improve overall symptoms and reduce pain. Patient education and a good doctor-patient relationship are an important part of care.

About 10 to 15% of people in the developed world are believed to be affected by IBS.It is estimated that 45% of people globally are affected by IBS.It is more common in South America and less common in Southeast Asia.It is twice as common in women as men and typically occurs before age 45.The condition appears to become less common with age.IBS does not affect life expectancy or lead to other serious diseases. The first description of the condition was in 1820 while the current term "irritable bowel syndrome" came into use in 1944.

Women are around two to three times more likely to be diagnosed with IBS and four to five times more likely to seek specialty care for it than men. These differences likely reflect a combination of both biological (sex) and social factors. Finally, sexual trauma is a major risk factor for IBS, with as many as 33% of those affected reporting such abuse. Because women are at higher risk of sexual abuse than men, sex-related risk of abuse may contribute to the higher rate of IBS in women

Cause of IBS

The risk of developing IBS increases six-fold after acute gastrointestinal infection. Post-infection, further risk factors are young age, prolonged fever, anxiety, and depression. Psychological factors, such as depression or anxiety, have not been shown to cause or influence the onset of IBS, but may play significant role in the persistence and perceived severity of symptoms.

1. **Stress :** It is believed that psychological stress may trigger IBS in predisposed individuals. The stress response in the body involves the hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic nervous system, both of which have been shown to operate abnormally in people with IBS. Psychiatric illness or anxiety precedes IBS symptoms in two-thirds of people with IBS, and psychological traits predispose previously healthy people to developing IBS after gastroenteritis.
2. **Post-infectious:** Approximately 10 percent of IBS cases are triggered by an acute gastroenteritis infection. Genetic defects relating to the innate immune system and epithelial barrier as well as high stress and anxiety levels appear to increase the risk of developing post-infectious IBS. Post-infectious IBS usually manifests itself as the diarrhea-predominant subtype. However, increased gut permeability is strongly associated with IBS regardless of whether IBS was initiated by an infection or not.A link between small intestinal bacterial overgrowth and tropical sprue has been proposed to be involved as a cause of post-infectious IBS.
3. **Bacteria:** Small intestinal bacterial overgrowth (SIBO) occurs with greater frequency in people who have been diagnosed with IBS compared to healthy controls. Symptoms of SIBO include bloating, abdominal pain, diarrhea or constipation among others. IBS may be the result of the immune system interacting abnormally with gut microbiota resulting in an abnormal cytokine signaling profile.
4. **Fungus:** The role of the gut mycobiota and especially of the abnormal proliferation of the yeast *Candida albicans* in some people with IBS is found.
5. **Protozoa:** Prevalence of protozoal infections is greater in industrialized countries (United States and Canada) in the 21st century. Evidences suggest that blastocystis-colonization occurs more commonly in IBS affected individuals and is a possible risk factor for developing IBS.*Dientamoebafragilis* has also been considered a possible organism to study, though it is also found in people without IBS.
6. **Vitamin D:** Vitamin D deficiency is more common in individuals affected by irritable bowel syndrome.Vitamin D is involved in regulating triggers for IBS including the gut microbiome, inflammatory processes and immune responses, as well as psychosocial factors.
7. **Genetics:** SCN5A mutations are found in a small number of people who have IBS, particularly the constipation-predominant variant (IBS-C). The resulting defect leads to disruption in bowel function, by affecting the Nav1.5 channel, in smooth muscle of the colon and pacemaker cells.

Mechanism

Genetic, environmental, and psychological factors seem to be important in the development of IBS. Studies have shown that IBS has a genetic component even though there is a predominant influence of environmental factors.

There is evidence that abnormalities occur in the gut flora of individuals who have IBS, such as reduced diversity, a decrease in bacteria belonging to the phylum *Bacteroidetes*, and an increase in those belonging to the phylum *Firmicutes*. The changes in gut flora are most profound in individuals who have diarrhoea predominant IBS. Antibodies against common components (namely flagellin) of the commensal gut flora are a common occurrence in IBS affected individuals.

Chronic low-grade inflammation commonly occurs in IBS affected individuals with abnormalities found including increased enterochromaffin cells, intraepithelial lymphocytes, and mast cells resulting in chronic immune-mediated inflammation of the gut mucosa. IBS has been reported in greater quantities in multigenerational families with IBS than in the regular population. It is believed that psychological stress can induce increased inflammation and thereby cause IBS to develop in predisposed individuals.

Signs and symptoms

- Abdominal pain or discomfort in association with frequents diarrhea or constipation and a change in bowel habits, these are experienced as acute attacks that subside within one day, but recurrent attacks are common.
- Urgency for bowel movements,
- A feeling of tenesmus or bloating.
- Some cases, the symptoms are relieved by bowel movements.
- People with IBS, more commonly than others, have gastroesophageal reflux, symptoms relating to the genitourinary system, chronic fatigue. syndrome, fibromyalgia, headache, backache, and psychiatric symptoms such as depression and anxiety.
- About a third of men and women who have IBS also report sexual dysfunction typically in the form of a reduction in libido.

Food intolerance

Symptoms in up to 90% of people are brought on by food intolerances.These often include fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs)

Diagnosis

No specific laboratory or imaging tests can diagnose irritable bowel syndrome. Diagnosis should be based on symptoms. Rome criteria are usually used to diagnose the cases of IBS. They allow the diagnosis to be based only on symptoms, but no criteria based solely on symptoms is sufficiently accurate to diagnose IBS. Worrisome features include onset at greater than 50 years of age, weight loss, blood in the stool, iron-deficiency anemia, or a family history of colon cancer, celiac disease, or inflammatory bowel disease.

Rome criteria

The Rome IV criteria for the diagnosis of irritable bowel syndrome require that patients have had recurrent abdominal pain on average at least 1 day per week during the previous 3 months that is associated with two or more of the following:

- Related to defecation (may be increased or unchanged by defecation)
- Associated with a change in stool frequency
- Associated with a change in stool form or appearance

The Rome IV criteria (May 2016) only require abdominal pain in defining this condition; "discomfort" is no longer a requirement owing to its non-specificity, and the recurrent abdominal pain.

Supporting symptoms include the following:

- Altered stool frequency
- Altered stool form
- Altered stool passage (straining and/or urgency)
- Mucorrhea
- Abdominal bloating or subjective distention

Physicians may choose to use one of these guidelines or may simply choose to rely on their own anecdotal experience with past patients. The algorithm may include additional tests to guard against misdiagnosis of other diseases as IBS. Such "red flag" symptoms may include weight loss, gastrointestinal bleeding, anemia, or nocturnal symptoms. However, red flag conditions may not always contribute to accuracy in diagnosis; for instance, as many as 31% of people with IBS have blood in their stool, many possibly from hemorrhoidal bleeding.

IBS Subtypes: IBS is classified into four subtypes such as-

1. IBS with predominant constipation (IBS-C)
2. IBS with predominant diarrhea (IBS-D)
3. IBS with mixed bowel habits (IBS-M)
4. IBS of unsubtyped.

One important change from Rome III, as noted above, is that the IBS subtype is explicitly based on the patient's reported predominant bowel habit on days with abnormal bowel movements, and not on an average of all days, which might include days with normal bowel habits. Abnormal bowel movements are classified using the Bristol stool form scale, which is described below. For clinical trials, or when appropriate in clinical settings, subjects should complete a 14-day bowel diary to most accurately categorize IBS subtypes. Bristol stool types 1 and 2 or types 6 and 7 are considered abnormal.

Bristol Stool Form Scale

The Bristol stool form scale (BSFS) was developed in the 1990s in the Bristol Royal Infirmary in England. The authors described seven types of stool, which are noted below:

- Type 1: Separate hard lumps, like nuts (hard to pass)
- Type 2: Sausage-shaped, but lumpy
- Type 3: Like a sausage but with cracks on its surface
- Type 4: Like a sausage or snake, smooth and soft
- Type 5: Soft blobs with clear cut edges (passed easily)
- Type 6: Fluffy pieces with ragged edges, a mushy stool
- Type 7: Watery, no solid pieces, entirely liquid

Stool types 1 and 2 as being associated with constipation, while stool types 6 and 7 were associated with diarrhea (and stool type 5 to some degree). Stool types 3 and 4 were considered normal stools. The BSFS is a convenient way for patients to describe their bowel habits, and is routinely used in clinical trials.

Differential diagnosis

Differential Diagnosis of IB

IBS with diarrhea

Dietary - lactose, sorbitol, fructose, caffeine, alcohol, fatty foods, fat substitutes, gas-producing foods

Infections- *Giardia* spp, *Amoeba* spp, HIV-related, bacterial overgrowth

IBD- Crohn's disease, ulcerative colitis, microscopic colitis

Drug toxicity- antibiotics, proton pump inhibitors, nonsteroidal anti-inflammatory drugs, ACE inhibitors, beta-blockers, chemotherapy

Malabsorption- celiac disease, bile acid-related

Other- ovarian cancer, endometriosis, colorectal cancer, hyperthyroidism, carcinoid, VIPoma, ischemic colitis

IBS with constipation

Dietary/mode of life- inadequate fiber, immobility

Neurologic- Parkinson's disease, multiple sclerosis, spinal cord injuries

Endocrine- diabetes, hypothyroidism, hypercalcemia

Drug toxicity- opiate analgesics, calcium-channel blockers, antidepressants, clonidine

Other - colorectal cancer, ovarian cancer, bowel obstruction, diverticular disease, endometriosis

Investigations

Investigations are performed to exclude other clinical conditions:

- Stool microscopy and culture (to exclude infectious conditions)
- Blood tests: Full blood examination, liver function tests, erythrocyte sedimentation rate, and serological testing for coeliac disease
- Abdominal ultrasound (to exclude gallstones and other biliary tract diseases)
- Endoscopy and biopsies (to exclude peptic ulcer disease, coeliac disease, inflammatory bowel disease, and malignancies)
- Hydrogen breath testing (to exclude fructose and lactose malabsorption)

Misdiagnosis

IBS patients are at increased risk of being given inappropriate surgeries such as appendectomy, cholecystectomy, and hysterectomy due to being misdiagnosed as other medical conditions.Some common examples of misdiagnosis include infectious diseases, coeliac disease,*Helicobacter pylori*, parasites (non-protozoal). The American College of Gastroenterology recommends all people with symptoms of IBS be tested for coeliac disease.

Bile acid malabsorption is also sometimes missed in people with diarrhea-predominant IBS. SeHCAT tests suggest around 30% of people with D-IBS have this condition, and most respond to bile acid sequestrants.

Chronic use of certain sedative-hypnotic drugs, especially the benzodiazepines, may cause irritable bowel-like symptoms that can lead to a misdiagnosis of irritable bowel syndrome.

Comorbidities

Several medical conditions, or comorbidities, appear with greater frequency in people with IBS.

- **Neurological/psychiatric:** IBS occurs in 51% of people with chronic fatigue syndrome and 49% of people with fibromyalgia, and psychiatric disorders occur in 94% of people with IBS.
- **Inflammatory bowel disease:** IBS may be a type of low-grade inflammatory bowel disease. Researchers have suggested IBS and IBD are interrelated diseases, noting that people with IBD experience IBS-like symptoms when their IBD is in remission.
- **Abdominal surgery:** People with IBS were at increased risk of having unnecessary gall bladder removal, abdominal, pelvic surgery etc.
- **Endometriosis:** There is a link between migraine headaches, IBS, and endometriosis.
- **Other chronic disorders:** Interstitial cystitis may be associated with other chronic pain syndromes, such as irritable bowel syndrome and fibromyalgia.

Inflammatory Bowel Disease

1. Ulcerative colitis and Crohn's disease are chronic inflammatory bowel diseases which pursue a protracted relapsing and remitting course, usually extending over years.
2. The diseases have many similarities and it is sometimes impossible to differentiate between them.
3. Ulcerative colitis only involves the colon
4. Crohn's disease can involve any part of the gastrointestinal tract from mouth to anus.
5. Crohn's disease appears to be very rare in the developing world yet ulcerative colitis, although still unusual, is becoming more common.
6. Life expectancy in patients with IBD is similar to that of the general population
7. Although many patients require surgery and admission to hospital for other reasons, the majority have an excellent work record and pursue a normal life.
8. Around 90% of ulcerative colitis patients have intermittent disease activity, whilst 10%, have continuous symptoms.

Comparison of ulcerative colitis and Crohn's disease

	Ulcerative colitis	Crohn's disease
Age group	Any	Any
Gender	M = F	M = F
Ethnic group	Any *HLA-DR103* associated with	Any; more common in Ashkenazi Jews
Genetic factors	severe disease	*CARD 15/NOD-2* mutations predispose
Risk factors	More co mmon in non-/ex-smokers Appendicectomy protects	More common in smokers
Anatomical distribution	Colon only; begins at anorectal margin with variable proximal extension	Any Part of gastrointestinal tract; perianal disease common; patchy distribution-'skip lesions'
Extraintestinal manifestations	Common	Common

Presentation	Bloody diarrhoea	Variable; pain, diarrhoea, weight loss all common
Histology	Inflammation limited to mucosa; crypt distortion, cryptitis, crypt abscesses, loss of goblet cells	Submucosal or transmural inflammation common; deep fissuring ulcers, fistulas; patchy changes; granulomas
Management	5-ASA; corticosteroids; azathioprine; colectomy is curative	Corticosteroids; methotrexate; biologic therapy (anti-TNF); nutritional therapy; surgery for complications is not curative

Pathophysiology

It is thought that IBD develops because of an abnormal host response to an environmental trigger in genetically susceptible individuals

↓

This causes inflammation of the intestine and release of inflammatory mediators, such as TNF,1L-12 and IL-23

↓

Which cause tissue damage.

↓

These mediators are targets for therapeutic intervention

↓

In both diseases the intestinal wall is infiltrated with acute and chronic inflammatory cells

↓

Inflammatory Bowel Disease

Factors associated with the development of inflammatory bowel disease

Genetic

- More common in Ashkenazi Jews
- 10% have a first-degree relative or at least one close relative with IBD
- High concordance between identical twins
- Both ulcerative colitis and Crohn's disease are associated with the HLA locus and the genes encoding MST-1 and ; IL-23
- Crohn's disease is associated with the *NOD2, ATGi6L1* and *IRGM* genes
- Ulcerative colitis is associated with the *ECM-1* gene
- Associated with autoimmune thyroiditis and SLE
- HLA-DR103 associated with severe ulcerative colitis
- Ulcerative colitis and Crohn's patients with HLA-B27 commonly develop ankylosing spondylitis

Environmental

- Ulcerative colitis is more common in non-smokers and ex-smokers
- Most Crohn's patients are smokers (relative risk = 3)
- Associated with low-residue, high refined sugar diet
- Appendicectomy protects against ulcerative colitis

Clinical features-Ulcerative colitis

1. The major symptom is bloody diarrhoea.
2. The first attack is usually the most severe and thereafter the disease is followed by relapses and remissions.
3. Emotional stress, intercurrent infection, gastroenteritis, antibiotics or NSAID therapy may all provoke a relapse.
4. Proctitis causes rectal bleeding and mucus discharge, sometimes accompanied by tenesmus.
5. Some patients pass frequent, small-volume fluid stools, while others are constipated and pass pellety stools.
6. Constitutional symptoms do not occur.

7. Proctosigmoiditis causes bloody diarrhoea with mucus.
8. Almost all patients are constitutionally well, but a small minority who have very active, limited disease develop fever, lethargy and abdominal discomfort.
9. Extensive colitis causes bloody diarrhoea with passage of mucus.
10. In severe cases anorexia, malaise, weight loss and abdominal pain occur, and the patient is toxic with fever, tachycardia and signs of peritoneal inflammation

Factors considered for disease severity assessment in ulcerative colitis

	Mild	Severe
Daily bowel frequency	< 4	> 6
Blood in stools	+/-	+++
Stool volume	< 200 g/24 hrs	> 400 g/24 hr.
Pulse	< 90 bpm	> 90 bpm
Temperature	Normal	> 37.8^{0}C, 2 days out of 4
Sigmoidoscopy	Normal or granular mucosa	Blood in lumen
Abdominal X-ray	Normal	Dilated bowel and/or mucosal islands
Haemoglobin	Normal	< 100 g/L (< 10 g/dL)
ESR	Normal	> 30 mm/hr
Serum albumin	> 35 g/L (> 3.5 g/dL)	< 30 g/L (< 3 g/dL)

Crohn's disease

1. The major symptoms are abdominal pain, diarrhoea and weight loss.
2. Real Crohn's disease may cause subacute or even acute intestinal obstruction.
3. The pain is often associated with diarrhoea which is usually watery and does not contain blood or mucus.
4. Almost all patients lose weight because they avoid food since eating provokes pain.
5. Weight loss may also be due to malabsorption, and some patients present with features of fat, protein or vitamin deficiencies.
6. Crohn's colitis presents in an identical manner to ulcerative colitis, but rectal sparing and the presence of perianal disease are features which favour a diagnosis of Crohn's disease.
7. Many patients present with symptoms of both small bowel and colonic disease.
8. A few have isolated perianal disease, vomiting from jejunal strictures or severe oral ulceration.

Physical examination

1. Physical examination often reveals
    - Evidence of weight loss
    - Anaemia
    - Glossitis
    - Angular stomatitis.
    - Abdominal tenderness most marked over the inflamed area.
    - An abdominal mass due to matted loops of thickened bowel or an intra- abdominal abscess may occur.
    - Perianal skin tags, fissures or fistulas are found in at least 50% of patients.

Investigations

Blood :

1. Full blood count
    - Anaemia resulting from bleeding or malabsorption of iron, folic acid or vitamin B12.
2. Serum albumin concentration falls as a consequence of protein-losing enteropathy, inflammatory disease or poor nutrition.
3. The ESR and CRP are elevated in exacerbations and in response to abscess formation.

Bacteriology :

4. Stool microscopy, culture and examination for *Clostridium difficile* toxin or for ova and cysts,
5. Blood cultures and serological tests should be performed to exclude infection.

Endoscopy :

6. Sigmoidoscopy with biopsy is an essential

investigation in all patients who present with diarrhoea.

7. Colonoscopy may show active inflammation with pseudopolyps or a complicating carcinoma.
8. Biopsies are confluent and most severe in the distal colon and rectum.
9. Capsule endoscopy is useful in the identification of small bowel inflammation, but should be avoided in the presence of strictures.

Radiology :

10. A plain abdominal X-ray is essential in the management of patients who present with severe active disease.
    - Dilatation of the colon, mucosal oedema ('thumb-printing') or evidence of perforation may be found.
    - In small bowel Crohn's disease there may be evidence of intestinal obstruction or displacement of bowel loops by a mass.
11. Barium enema is a less sensitive investigation than colonoscopy in patients with colitis and is now little used.
    - Contrast studies of the small bowel can be helpful in the investigation of Crohn's disease; affected areas are narrowed and ulcerated, and multiple strictures are common

MRI scans :

12. MRI scans provide comparable information without radiation exposure or risking small bowel obstruction and are useful in delineating pelvic or perineal involvement.

Ultrasound :

13. Ultrasound may identify thickened small bowel loops and abscess development in Crohn' s disease.

MCQs

1. Duodenal Ulcers occur most often in the
   a. First portion of the duodenum (95%), with 90% located within 3 cm ofthe pylorus and they are usually ≤ 1 cm in diameter but can occasionallyreach 3–6 cm (giant ulcer).
   b. Second portion of the duodenum (95%), with 90% located within 4 cmof the pylorus and they are usually ≤ 2 cm in diameter but canoccasionally reach 4–8 cm (giant ulcer).
   c. First portion of the duodenum (95%), with 90% located within 2 cm reach 2–5 cm (giant ulcer).
   d. Second portion of the duodenum (95%), with 90% located within 1 cmof the pylorus and they are usually ≤ 3 cm in diameter but canoccasionally reach 6–8 cm (giant ulcer).
2. The Rome criteria specify that symptoms that characterize IBS must have been present for the previous 3 months. Which of the following is one of the Rome criteria for diagnosing irritable bowel syndrome (IBS)?
   a. Flatulence
   b. Nausea
   c. Abdominal pain 3 or more days per week
   d. Pain with defecation
3. During physical examination of a patient with suspected IBS, which abdominal location is most likely to be tender to palpation?
   a. Left upper quadrant
   b. Right upper quadrant
   c. Left lower quadrant
   d. Right lower quadrant
4. Which of the following infections has been associated with an increased prevalence of IBS?
   a. Giardia lamblia b. Escherichia coli
   c. Shigella d. Salmonella
5. Which of the following is a symptom consistent with a diagnosis of IBS?
   a. Painless diarrhea
   b. Fever
   c. Postprandial urgency
   d. Steatorrhea
6. Which of the following is not recognized as a symptom that supports the diagnosis of IBS according to the Rome criteria?
   a. Altered stool frequency
   b. Mucorrhea
   c. Abdominal bloating or subjective distention
   d. Frequent nausea

7. Which of the following symptoms indicates a need for laboratory testing or diagnostic imaging in patients with IBS younger than 50 years?
   a. Iron deficiency anemia
   b. Abdominal pain
   c. Amenorrhea
   d. Hypokalemia
8. Which of the following is accurate about endoscopic findings in PUD and related conditions?
   a. Most gastric ulcers tend to occur at the junction of the fundus and antrum, along the lesser curvature
   b. Most duodenal ulcers are found in the second and third parts of the duodenum
   c. Malignant ulcers tend to have a smooth, regular, rounded edge with a flat, smooth base and surrounding mucosa that shows radiating folds
   d. The most common finding in individuals undergoing endoscopy for dyspepsia is peptic ulcer

Answer

1.	a	2.	d	3.	c
4.	a	5.	c	6.	d
7.	a	8.	a		

CHAPTER

9

Mutrakricchra

Introduction

Painful discharge of urine, strangury; a class of urinary afflictions (of which 8 kinds are enumerated). *Mūtra Kṛcchra* is an indisposition resulting from various produces leading to painful micturation. In this indisposition the patient passes frequently small quantity of urine with difficulty, pain and burning sensation. The urine could also contain blood.

मूत्रकृच्छ्र हेतु (Mūtrakṛcchra Hetu)

> व्यायामतीक्ष्णौषधरुक्षमद्य प्रसङ्गनित्यद्रुतपृष्ठयानात् ।
> आनूपमत्स्याध्यशनादजीर्णात् स्युर्मूत्रकृच्छ्राणि नृणामिहाष्टौ ॥
> *C.S.Ci. 26/32*

Causative factors of Mūtrakṛcchra are as follows : Excessive physical exercises, consumption of irritant drugs, rough food, regular intake of wine, riding over the back of the fast moving animals, eating meat of marshy land animals & fish; eating during indigestion and indigestion.

Due to above *Hetus* eight varieties of *Mūtrakṛcchra* develops.

> पृथङ्मलाः स्वैः कुपिता निदानैः सर्वेऽथवा कोपमुपेत्य बस्तौ ।
> मूत्रस्य मार्गं परिपीडयन्ति यदा तदा मूत्रयतीह कृच्छ्रात् ॥
> *C.S.Ci. 26/33*

Due to above etiological factors *Doṣās* vitiated separately by their respective causes or all together get localised in the *Basti* and afflicts its passage leading to genesis of *Mūtrakṛcchra.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi Doṣās*
↓
Get localised in the *Basti* region
↓
Afflicts its passage due to amalgamation of *doṣa* evam *dūṣya*
↓
Mūtrakṛcchra

मूत्रकृच्छ्र संप्राप्ति घटक (Samprāpti Ghaṭaka of Mūtrakṛcchra)

Doṣa	:	*Tridoṣaja (Vātapradhāna)* *Vāta → Apāna* *Pitta → Pācaka* *Kapha → Kledaka*
Dūṣya	:	*Dhātu → Rasa, Rakta, Śukra* *Updhātu→* *Śārīrika Mala → Mūtra, Puriṣa* *Dhātu Mala → Kapha, Pitta*
Agni	:	*Jaṭharāgni, Dhātvāgni*
Agniduṣṭi	:	*Mandāgni*
Āma	:	*Āgnijanya*
Srotas	:	*Mūtravaha, Purīṣavaha*
Srotoduṣṭi	:	*Saṅga, Sirāgranthi*
Adhiṣṭhāna	:	*Basti*
Udbhavasthāna	:	*Pakvāśayottha & Āmāśayottha*
Sancārasthāna	:	*Mūtravāhinyaḥ*
Vyaktasthāna	:	*Mūtravaha Sroto Avayava, Basti, Mehana*
Svabhāva	:	*Cirakāri & Āśukāri*
Prabhāva	:	*Kṛcchrasādhya*
Rogamārga	:	*Madhyama*

रोग भेद (Roga Bheda)

> वातेन पित्तेन कफेन सर्वैस्तथाऽभिघातैः शकृदश्मरीभ्याम् ।
> तथाऽपरः शर्करया सुकष्टो मूत्रोपघातः कथितोऽष्टमस्तु ॥
> *S.S.Utt. 59/3*

Mūtrakṛcchra is classified into eight varieties.

1. *Vātaja*
2. *Pittaja*
3. *Kaphaja*
4. *Sannipātaja*
5. *Abhighātaja*
6. *Śakṛdaja*
7. *Aśmarīja*
8. *Śarkarāja.*

Caraka classified into eight kinds namely :

1. *Vātaja*
2. *Pittaja*
3. *Kaphaja*
4. *Sannipātaja*
5. *Aśmarīja*
6. *Śarkarāja*
7. *Śukraja*
8. *Kṣataja.*

वातज मूत्रकृच्छ्र (Vātaja Mūtrakṛcchra)

तीव्रा रुजो वङ्क्षणबस्तिमेढ्रे स्वल्पं मुहुर्मुत्रयतीह वातात् ।
C.S.Ci. 26/34

अल्पमल्पं समुत्पीड्य शुष्कमेहनबस्तिभिः ।
फलद्भिरिव कृच्छ्रेण वाताघातेन मेहति ॥
S.S.Utt. 59/4

Vātaja Mūtrakṛcchra is characterised by elimination of little quantity of urine oftenly with difficulty associated with excruciating pain in the groins, scrotum, penis and urinary bladder.

पित्तज मूत्रकृच्छ्र (Pittaja Mūtrakṛcchra)

आनूपमत्स्याध्यशनादजीर्णात् स्युर्मूत्रकृच्छ्राणि नृणामिहाष्टौ ॥
C.S.Ci. 26/34

हारिद्रमुष्णं रक्तं वा मुष्कमेहनबस्तिभिः ।
अग्निना दह्यमानाभैः पित्ताघातेन मेहति ॥
S.S.Utt. 59/5

Pittaja Mūtrakṛcchra is characterised by elimination of yellowish, hot & red urine associated with burning sensation in the scrotum, penis & bladder, passes urine very frequently with difficulty & pain.

कफज मूत्रकृच्छ्र (Kaphaja Mūtrakṛcchra)

बस्तेः सलिङ्गस्य गुरुत्वशोथौ मूत्रं सपिच्छं कफमूत्रकृच्छ्रे ।
C.S.Ci. 26/35

स्निग्धं शुक्लमनुष्णञ्च मुष्कमेहनबस्तिभिः ।
संहृष्टरोमा गुरुभिः श्लेष्माघातेन मेहति ॥
S.S.Utt. 59/6

Kaphaja Mūtrakṛcchra is characterised by elimination of unctous, white, little warm, slimy urine associated with heaviness & swelling in bladder & penis, feeling of heaviness in scrotum, penis & bladder.

सन्निपातज मूत्रकृच्छ्र (Sannipātaja Mūtrakṛcchra)

सर्वाणि रुपाणि तु सन्निपाताद् भवन्ति तत् कृच्छ्रतमं हि कृच्छ्रम् ॥ *C.S.Ci. 26/35*

दाहशीतरुजाविष्टो नानावर्णं मुहुर्मुहुः ।
ताम्यमानस्तु कृच्छ्रेण सन्निपातेन मेहति ॥
S.S.Utt. 59/7

Sannipātaja Mūtrakṛcchra is characterised by elimination of multi coloured urine, oftenly associated with burning sensation or cold, pain & loss of consciousness. Appearance of all the above signs & symptoms are observed. It is very difficult for management.

अश्मरीज मूत्रकृच्छ्र (Aśmarīja Mūtrakṛcchra)

विशोषयेद्वस्तिगतं सशुक्रं मूत्रं सपित्तं पवनः कफं वा ।
यदा तदाऽश्मर्युपजायते तु क्रमेण पित्तेष्विव रोचना गोः ॥
कदम्बपुष्पाकृतिरश्मतुल्या श्लक्ष्णा त्रिपुट्यप्यथवाऽपि मृद्वी ।
मूत्रस्य चेन्मार्गमुपैति रुद्ध्वा मूत्रं रुजं तस्य करोति बस्तौ ॥
ससेवनीमेहनबस्तिशूलं विशीर्णधारं च करोति मूत्रम् ।
मृद्नाति मेढ्रं स तु वेदनार्तो मुहुः शकृन्मुञ्चति मेहते च ॥
क्षोभात् क्षते मूत्रयतीह सासृक् तस्याः सुखं मेहति च व्यपायात् ।
C.S.Ci. 26/36-39

Aggravated *Vāta* dries up the semen, urine, *Pitta* & *Kapha* located in the urinary bladder leading to development of *Aśmarī* (stones) like gallstone in cow.

It is classified into four varieties namely :

1. *Vātaja*
2. *Pittaja*
3. *Kaphaja*
4. *Śukraja.*

Vātaja Aśmarī appears like the flower of *Kadamba* & is of *tripuṭi* (having three layers).

Pittaja Aśmarī appears like stone & which is smooth.

Kaphaja & *Śukraja Aśmarī* are soft.

Obstruction to urinary passage takes place due to *Aśmari* as a result patient suffers from pain in the urinary bladder, perineum, phallus & hypogastric region associated with splitting of urine while micturition. Due to agonising pain, patient squeezes penis and frequently passes urine & stool. If the urinary passage gets injured by *Aśmarī* leading to elimination of urine mixed with blood. Patient passes urine easily after eliminating *Aśmarī*. *Suśruta* says symptoms of *Aśmarī* & *Sarkarā* are similar.

शर्कराज मूत्रकृच्छ्र (Śarkarāja Mūtrakṛcchra)

एषाऽश्मरी मारुतभिन्नमूर्तिः स्याच्छर्करा मूत्रपथात् क्षरन्ती ॥
C.S.Ci. 26/39

पच्यमानस्य पित्तेन भिद्यमानस्य वायुना ।
श्लेष्मणोऽवयवा भिन्नाः शर्करा इति संज्ञिताः ॥
हृत्पीडा वेपथुः शूलं कुक्षौ वह्निः सुदुर्बलः ।
ताभिर्भवति मूर्च्छा च मूत्राघातश्च दारुणः ॥
मूत्रवेगनिरस्तासु तासु शाम्यति वेदना ।
यावदन्या पुनर्नैति गुडिका स्रोतसो मुखम् ॥
शर्करासम्भवस्यैतन्मूत्राघातस्य लक्षणम् ।
S.S.Utt. 59/12-15

When *Aśmarī* becomes disintegrated into small particles by *Vāta* & these come out from urinary passage is known as *Śarkarā.*

Aśmarī undergoing processing by *Pitta,* broken into many pieces by *Vāta,* separated into parts by *Kapha* & the same comes out of urine is known as *Śarkarā.* It is characterised by pain in the region of heart, shivering, abdominal pain, sluggishness of digestive fire, fainting & severe dysuria. Pain subsides after elimination of urine & appears again when the passage is blocked by stone gravel.

शुक्रज मूत्रकृच्छ्र (Śukraja Mūtrakṛcchra)

रेतोऽभिघाताभिहतस्य पुंसः प्रवर्तते यस्य तु मूत्रकृच्छ्रम् ।
स्याद्वेदना वङ्क्षणवस्तिमेढ्रे तस्यातिशूलं वुष्मातिवृत्ते ॥
शुक्रेण संरुद्धगतिप्रवाहो मूत्रं स कृच्छ्रेण विमुञ्चतीह ।
तमण्डयोः स्तव्धमिति ब्रुवन्ति रेतोऽभिघातात् प्रवदन्ति कृच्छ्रम् ॥
शुक्रं मलाश्चैव पृथक् पृथग्वा मूत्राशयस्थाः प्रतिवारयन्ति ।
तद्व्याहतं मेहनवस्तिशूलं मूत्रं सशुक्रं कुरुते विबद्धम् ॥
स्तव्धश्च शूनो भृशवेदनश्च तुद्येत बस्तिर्वृषणौ च तस्य ।
C.S.Ci. 26/40-43

Śukraja Mūtrakṛcchra is characterised by pain in the groin, urinary bladder & penis, testicles become enlarged & painful. Due to obstruction to urinary flow by semen patient passes urine with difficulty and stiffness in testicles.

All the *Malās (Doṣās)* located in the urinary bladder obstruct the passage of semen causing pain in the penis & urinary bladder. Patient suffers difficulty in micturition & ejaculation of semen associated with stiffness, swelling, excessive pricking pain in the region of urinary bladder & testicles.

क्षतज मूत्रकृच्छ्र (Kṣataja Mūtrakṛcchra)

क्षताभिघातात् क्षतजं क्षयाद्वा प्रकोपितं बस्तिगतं विबद्धम् ॥
तीव्रार्ति मूत्रेण सहाश्मरीत्वमा याति तस्मिन्नतिसंचिते च ।
आध्माततां विन्दति गौरवं च बस्तेर्लघुत्वं च विनिःसृतेऽस्मिन् ॥
C.S.Ci. 26/43-44

Blood present in the urinary bladder gets vitiated due to trauma, *Śukra Kṣaya,* it causes obstruction & excruciating pain in the urinary bladder. Excessively accumulated vitiated blood forms calculus in association with urine, which causes distension & heaviness in the urinary bladder and which is relieved after the calculus passes out.

शकृतज मूत्रकृच्छ्र (Śakṛtaja Mūtrakṛcchra)

शकृतस्तु प्रतीघाताद्वायुर्विगुणतां गतः ।
आध्मानञ्च सशूलञ्च मूत्रसङ्गं करोति हि ॥
S.S.Utt. 59/9-10

Vāta getting aggravated by the retention of stool, causes flatulence, painful micturition & retention of urine.

शकृतज मूत्रकृच्छ्र चिकित्सा (Śakṛtaja Mūtrakṛcchra Cikitsā)

मूत्रकृच्छ्रे शकृज्जाते कार्या वातहरी क्रिया ।
स्वेदावगाहावभ्यङ्गबस्तिचूर्णक्रियास्तथा ॥
S.S.Utt. 59/26

The therapies alleviating *vāta* like sudation, tubbath, anointing, enema and consumption of the powders.

उपशय/पथ्य (Upaśaya/Pathya)

The following products are very much beneficial for the patient suffering from *Mūtrakṛchra*: old red coloured *Śāli* rice, *Kṣāra* products, barley, hot products, buttermilk, milk, curd of cow, meat of wild animals and birds, water of *Mudga* pulse, sugar, fruit of *Kūṣmāṇḍa*, leaves of *Paṭola*, wild ginger, *Gokṣura*, *Ghṛtakumārī*, betel nut, date fruit, pulp of unripe coconut, cucumber, smaller cardamom, cold edibles, fresh water of river and camphor. (B.R. 34/67-69)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

व्यायामसंधारणशुष्करूक्षपिष्टान्नवातार्ककशव्यवायान् ।
खर्जूरशालूककपित्थजम्बूबिसं कषायं न रसं भजेत ॥
C.S.Ci. 26/76

Mūtrakṛcchra patients should avoid exercise, suppression of natural urges, dry & rough food substances, & flour preparations, exposure to wind, sexual intercourse, *Kharjūra*, lotus root, *Kapittha*, *Jambū*, lotus stem & substances having astringent taste.

The following measures are prohibited to the patient of *Mūtrakṛcchra*: hard labour, suppression of natural urges, intercourse and riding elephants or horses. The patient should also avoid taking incompatible meals, excessive or very less amount of food, betel nuts, fishes, salty edibles, ginger, oily edibles, *Kalka* of sesame seeds, asafoetida, sesame oil, mustard oil, *Māṣa* or foods prepared from it, the *Karīraphalā* or the like bitter, hot, unctuous and citrus edibles. (B.R. 34/70-71)

MCQs

1. Types of *Mutrakrichra* as per *Ashtanga Hridaya*
   a. 20 b. 13
   c. 4 d. 12
2. *Dahashitarujavisto feature* seen in
   a. *Vataja Mutrakrichra*
   b. *Pittaja Mutrakrichra*
   c. *Kaphaja Mutrakrichra*
   d. *Sannipatika Mutrakrichra*
3. *Nityadrutaprishtayana* is the *nidana* of which disease as per *Madavanidana*
   a. *Mutrakrichra* b. *Mutraghata*
   c. *Asmari* d. *Gulma*
4. Types of *Mutrakrichra* as per *Madavanidana*
   a. *4* b. *20*
   c. *13* d. *8*
5. *What is the prognosis of Tridoshaja Mutrakrichra*
   a. *Sadya* b. *Krichrasadya*
   c. *Krichratama* d. *Asadya*
6. The features of *Mutrakrichra* is seen in *Salyaabhighataja Mutrakrichra*
   a. *Vataja Mutrakrchra*
   b. *Pittaja Mutrakrchra*
   c. *Asmari Mutrakrchra*
   d. *Sannipatika Mutrakrchra*
7. *Brisadarunam* is the prognosis of which type of *Mutrakrchra*
   a. *Asmari Mutrakrchra*
   b. *Sannipatika Mutrakrchra*
   c. *Salyaabhighataja Mutrakrchra*
   d. *Sukraja Mutrakrchra*
8. *Vasthimehanasoola seen in*
   a. *Asmari Mutrakrchra*
   b. *Sannipatika Mutrakrchra*
   c. *Sukraja Mutrakrchra*
   d. *Salyaabhighataja Mutrakrchra*

Answer

1.	c	2.	d	3.	a
4.	d	5.	c	6.	a
7.	c	8.	c		

CHAPTER
10

Mutraghata

मूत्राघात (Mūtrāghāta)

Introduction

Urinary disease (of which 12 or 13 kinds). Retention of urine in *Mūtrāghāta* patient is unable to pass the urine or passes a little urine with difficulty even though his bladder is full of urine. Anuria. Retention of urine.

मूत्राघात रोग भेद (Mūtrāghāta Roga Bheda)

वातकुण्डलिकाऽष्ठीला वातबस्तिस्तथैव च।
मूत्रातीतः सजङ्गरो मूत्रोत्सङ्गः क्षयस्तथा॥
मूत्रग्रन्थिर्मूत्रशुक्रमुष्णवातस्तथैव च।
मूत्रौकसादौ द्वौ चापि रोगा द्वादश कीर्त्तिताः॥
S.S.Utt. 58/3-4

Twelve varieties of Mūtrāghāta described are as follows:-

i) *Vātakuṇḍalikā,* ii) *Vātāṣṭhīlā,* iii) *Vātabasti,* iv) *Mūtrātīta,* v) *Mūtra Jaṭhara,* vi) *Mūtrotsaṅga,* vii) *Mūtrakṣaya,* viii) *Mūtragranthi,* ix) *Mūtraśukra,* x) *Uṣṇavāta,* xi) *Pittaja Mūtraukasāda* and xii) *Kaphaja Mūtraukasāda.*

वातकुण्डलिका (Vātakuṇḍalikā)

रौक्ष्याद्वेगविघाताद्वा वायुरन्तरमाश्रितः।
मूत्रं चरति सङ्गृह्य विगुणः कुण्डलीकृतः॥
सृजेदल्याल्पमथवा सरुजस्कं शनैः शनैः।
वातकुण्डलिकां तं तु व्याधिं विद्यात् सुदारुणम्॥
S.S.Utt. 58/5-6

Indulgence in dry foods, suppression of natural urges etc. causing aggravation of *Vāta* inside the urinary bladder, begins to move inside the urinary bladder along with urine in a circular manner and manifest *Vātakuṇḍalikā.* Patient passes urine oftenly, in small quantity associated with pain is known as *Vātakuṇḍalikā* which is dreadful.

वाताष्ठीला (Vātāṣṭhīla)

शकृन्मार्गस्य बस्तेश्च वायुरन्तरमाश्रितः।
अष्ठीलावद् घनं ग्रन्थिं करोत्यचलमुन्नतम्॥
विण्मूत्रानिलसङ्गश्च तत्राध्मानञ्च जायते।
वेदना च परा बस्तौ वाताष्ठीलेति तां विदुः॥
S.S.Utt. 58/7-8

Aggravated *Vāta* gets localised inside the passage of faeces & urinary bladder produces a hard tumour like cobblers stone, which is immovable & bulged up causing obstruction to faeces, urine & flatus, flatulence, severe pain in the urinary bladder are the characteristic features of *Vātāṣṭhīla.*

वातबस्ति (Vātabasti)

वेगं विधारयेद्यस्तु मूत्रस्याकुशलो नरः।
निरुणद्धि मुखं तस्य बस्तेर्बस्तिगतोऽनिलः॥
मूत्रसङ्गो भवेत्तेन बस्तिकुक्षिनिपीडितः।
वातबस्तिः स विज्ञेयो व्याधिः कृच्छ्रप्रसाधनः॥
S.S.Utt. 58/9-10

Suppression of natural urges by a person causes aggravation of *Vāta* in the urinary bladder and blocks the mouth of the bladder leading to retention of urine, pain in the bladder & abdomen are the characteristic features of *Vātabasti* which is diffifult to treat.

मूत्रातीत (Mūtrātīta)

वेगं सन्धार्य्य मूत्रस्य यो भूयः स्त्रष्टुमिच्छति।
तस्य नाभ्येति यदि वा कथञ्चित्सम्प्रवर्त्तते॥
प्रवाहतो मन्दरुजमल्पमल्पं पुनः पुनः।
मूत्रातीतन्तु तं विद्यान्मूत्रवेगविघातजम्॥
S.S.Utt. 58/11-12

One who has suppressed the urge of micturition for a long time and then desires to urinate it, then the urine does not come out or comes out in little quantity after straining associated with mild pain. Patient passes little quantity of urine oftenly. These are the characteristic features of *Mūtrātīta* produced by suppression of urge of urination.

मूत्रजठर (Mūtrajaṭhara)

मूत्रस्य विहते वेगे तदुदावर्त्तहेतुना ।
अपानः कुपितो वायुरुदरं पूरयेद् भृशम् ॥
नाभेरधस्तादाध्मानं जनयेत्तीव्रवेदनम् ।
तं मूत्रजठरं विद्यादधःस्त्रोतोनिरोधनम् ॥
S.S.Utt. 58/13-14

Due to suppression of urge of micturition, *Apānavāta* become aggravated and moves in upward direction, accumulates in the abdomen, causing distension below the umbilicus, severe pain and obstruction to lower channels are the characteristic features of *Mūtrajaṭhara.*

मूत्रोत्सङ्ग (Mūtrotsaṅga)

बस्तौ वाऽप्यथवा नाले मणौ वा यस्य देहिनः ।
मूत्रं प्रवृत्तं सज्जेत सरक्तं वा प्रवाहतः ॥
स्त्रवेच्छनैरल्पमल्पं सरुजं वाऽथ नीरुजम् ।
विगुणानिलजो व्याधिः समूत्रोत्सङ्गसंज्ञितः ॥
S.S.Utt. 58/15-16

Patient suffers from obstruction to the flow of urine either in the bladder, urethra or glans penis. Patient passes urine mixed with blood after straining, slowly, little in quantity, with or without pain are the characteristic features of Mūtrasaṅga & it manifest due to aggravated Vāta.

मूत्रक्षय (Mūtrakṣaya)

रूक्षस्य क्लान्तदेहस्य बस्तिस्थौ पित्तमारुतौ ।
सदाहवेदनं कृच्छं कुर्य्यातां मूत्रसङ्क्षयम् ॥
S.S.Utt. 58/17

One who is habituated to dry foods & physical fatigue causing aggravation of *Pitta* & *Vāta* in the urinary bladder and manifest burning sensation & pain in the urinary bladder associated with difficulty in micturition are the characteristic features of *Mūtrakṣaya.*

मूत्रग्रन्थि (Mūtragranthi)

अभ्यन्तरे बस्तिमुखे वृत्तोऽल्पः स्थिर एव च ।
वेदनावानति सदा मूत्रमार्गनिरोधनः ॥
जायते सहसा यस्य ग्रन्थिरश्मरिलक्षणः ।
स मूत्रग्रन्थिरित्येवमुच्यते वेदनाऽऽदिभिः ॥
S.S.Utt. 58/18-19

It is characterised by development of small, round & immovable tumour inside the urinary bladder associated with severe pain, obstruction to urinary passage and other features of *Aśmarī.*

मूत्रशुक्र (Mūtra Śukra)

प्रत्युपस्थितमूत्रस्तु मैथुनं योऽभिनन्दति ।
तस्य मूत्रयुतं रेतः सहसा सम्प्रवर्तते ॥
पुरस्ताद्वाऽपि मूत्रस्य पश्चाद्वाऽपि कदाचन ।
भस्मोदकप्रतीकाशं मूत्रशुक्रं तदुच्यते ॥
S.S.Utt. 58/20-21

One who performs sexual intercourse during the urge of urination, the semen mixed with urine comes out either before or after micturition and urine appears like solution of ash are the characteristic features of *Mūtraśukra.*

उष्णवात (Uṣṇavāta)

व्यायामाध्वातपैः पित्तं बस्तिं प्राप्यानिलावृतम् ।
बस्तिं मेढ्रं गुदञ्चैव प्रदहन् स्त्रावयेदधः ॥
मूत्रं हारिद्रमथवा सरक्तं रक्तमेव वा ।
कृच्छ्रात् प्रवर्तते जन्तोरुष्णवातं वदन्ति तम् ॥
S.S.Utt. 58/22-23

Excessive physical exercise, walking long distances & exposure to sun causes aggravation of *Pitta* and *Vāta* inside the urinary bladder and manifest burning sensation in the bladder, penis & rectum; urine becomes yellow, mixed with blood or only blood and comes out with difficulty are the characteristic feature of *Uṣṇavāta.*

पित्तज मूत्रौकसाद (Pittaja Mūtraukasāda)

विशदं पीतकं मूत्रं सदाहं बहलं तथा ।
शुष्कं भवति यच्चापि रोचनाचूर्णसन्निभम् ॥
मूत्रौकसादं तं विद्याद्रोगं पित्तकृतं बुधः ।
पिच्छिलं संहतं श्वेतं तथा कृच्छ्रप्रवर्त्तनम् ॥
S.S.Utt. 58/24-25

Patient passes urine which is non-slimy, yellow, thick and dry associated with burning sensation. Urine resemble like powder of oxgall (yellow) in colour are the characteristic features of *Pittaja Mūtraukasāda.*

कफज मूत्रौकसाद (Kaphaja Mūtraukasāda)

शुष्कं भवति यच्चापि शङ्खचूर्णप्रपाण्डुरम् ।

मूत्रौकसादं तं विद्यादामयं द्वादशं कफात् ॥
S.S.Utt. 58/26

Patient passes urine which is dry, thick, whitish & eliminates with difficulty. Urine appears like powder of conch or too white are the characteristic features of *Kaphaja Mūtraukasāda.*

विड् विधात (Viḍ Vighāta)

रूक्षदुर्बलयोर्वातादुदावर्तं शकृद्यथा ॥
मूत्रस्रोतोऽनुपर्येति संसृष्टं शकृता तदा ।
मूत्रं विट्तुल्यगन्धं स्याद्विड्विघातं तमादिशेत् ॥
A.H.Ni. 9/33-34

Vāta moves upwards in those individuals who is suffering from roughness & debilitation causing faecal matter into the channels of urine produces difficulty in micturition & urine assumes the smell of faeces are the characteristic features of *Viḍ Vighāta.*

Defferentiate Mūtrakṛcchra and Mūtrāghāta

S.No.	Mūtrakṛcchra	Mūtrāghāta
1	*Mūtrakṛcchra* is a condition develops due to inflammation, hormonal, Psychogenic conditions, trauma, Malformations, neoplasms and infections and it may be termed as Dysuria,	*Mūtrāghāta* is a condition develops due to obstruction in the Urinary Tract of vivid origin like Neurogenic disturbances of the urinary bladder & Urinary outflow obstruction
2	*Mūtrakṛcchra* classified into eight kinds namely: 1. *Vātaja Mūtrakṛcchra* 2. *Pittaja Mūtrakṛcchra* 3. *Kaphaja Mūtrakṛcchra* 4. *Sannipātaja Mūtrakṛcchra* 5. *Aśmarīja Mūtrakṛcchra* 6. *Śarkarāja Mūtrakṛcchra* 7. *Śukraja Mūtrakṛcchra* 8. *Kṣataja. Mūtrakṛcchra*	Thirteen varieties of *Mūtrāghāta* described are as follows:– *1. Vātakuṇḍalikā* 2. *Vātāṣṭhīlā,* *3. Vātabasti* *4. Mūtrātīta, 5. Mūtra Jaṭhara* *6. Mūtrotsaṅga, 7. Mūtrakṣaya,* *8. Mūtragranthi,* 9. *Mūtraśukra,* 10. *Uṣṇavāta,* 11. M*ūtrasāda* and *12. Viḍvighāta* 13. *Vastikuṇḍala*

उपशय/पथ्य (Upaśaya/Pathya)

Ksaudrārdha Sarpi, Balādighṛta, Mahābalādi Ghṛta.

The following measures are very helpful in treating the indisposition of *Mūtrāghāta:-* Massaging through oil, intake of oily materials, *Basti* (enema) and *Virecana* (purgation) therapy, sweating, *Uttara Basti* and water sports. Also helpful is intake of old red variety of rice, meat of the birds and animals belonging to the deserts, liquor, buttermilk, milk, curd, cooked *Urada* pulse, old fruits of *Kūṣmāṇḍa* and *Paṭola*, ginger and *Tintiḍikā*, pulp of the stone of palm fruit, *Haraḍa*, unripe coconut fruits, betel nuts, head (top portion) of the date, coconut and palm trees. (B.R. 30/50-52)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures are harmful in treating the indisposition of *Mūtrāghāta* : incompatible meals, excess exercise, continuing in the cold, intercourse, Suppression of the natural urges, taking the fruit *Karīra* and vomiting. (B.R. 30/53)

MCQs

1. *Asmaritulyaruk* is seen in
   a. *Mutrajatara* b. *Mutragranti*
   c. *Mutrakundalika* d. *Mutrakshaya*
2. *Dosha* predominance of *Mutraksaya*
   a. *Tridoshaja* b. *Vatakaphaja*
   c. *Pitta maruta* d. *Kaphapaithika*
3. *Dustara* & *Dustaratara* are the types of
   a. *Vatabasti* b. *Mutraatitha*
   c. *Mutrasukra* d. *Mutrakshaya*
4. Which among the following is not mentioned by *Susruta*
   a. *Mutraatitha* b. *Mutrasukra*
   c. *Mutrakshaya* d. *Vid vighata*
5. *MandaPravritti* of urine is seen in
   a. *Mutra-Atita* b. *Mutra-Jathara*
   c. *Mutroukasada* d. *Ushnavata*
6. *Basti Adhmana* and severe pain is seen in
   a. *Mutra-Atita* b. *Mutra-Jathara*
   c. *Mutrou Kasaada* d. *Ushnavata*

7. *Mutra* resembling *Rocana-Curna* is seen in
   a. *Mutra-*Atita b. *Mutra-Jadhara*
   c. *Mutou Kasaada* d. *Ushnavata*
8. *Vyayama* and *Adhvagamana* cause
   a. *Mutra-Atita* b. *Mutra-Jathara*
   c. *Mutou Kasaada* d. *Ushnavata*
9. *Bhasmodakapratheekasamutram* is the feature of
   a. Ushnavata *b. Mutrasukra*
   c. Mutrasada *d. Mutragranthi*
10. Bindumbindumsravatyapi, sastravishopama are the features of
    a. *Mutraatitha* b. *Mutrasukra*
    c. *Mutrakshaya* d. *Vasthikundala*
11. Srujedalpalpamathava sarujaskam shanai shanai are features of
    a. *Mutraatitha* b. *Mutrasukra*
    c. *Mutrakshaya* d. *Vatakundalika*

Answer

1.	b	2.	c	3.	a
4.	d	5.	a	6.	b
7.	c	8.	d	9.	b
10.	d	11.	d		

CHAPTER

11

Common Urinary Disease

Urinary tract infection (UTI)

1. In health bacterial colonisation is confined to the lower end of the urethra and the remainder of the urinary tract is sterile.
2. UTI is the most common bacterial infection managed in general medical practice and accounts for 1-3% of consultation.
3. Up to 50% of women have a UTI at some time.
4. The prevalence of UTI in women is about 3% at the age of 20, increasing by about 1% in each subsequent decade.
5. In male UTI is uncommon, except in the first year of life and in men over 60, in whom urinary tract obstruction due to prostatic hypertrophy may occur.

Aetiology and risk factors

1. Urine is an excellent culture medium for bacteria; in addition, the urothelium of susceptible persons may have more receptors to which virulent strains of *Escherechia coli* become adherent.
2. In women, the ascent of organ into the bladder is easier than in men; the urethra is shorter and absence of bactericidal prostatic secretions may be relevant.
3. Sexual intercourse may cause minor urethral trauma and transfer bacteria from the perineum into the bladder.
4. Instrumentation of the bladder may also introduce organisms.
5. Multiplication of organisms is depends on a number of factors, including the size of the inoculum and virulence of the bacteria.

Conditions which predispose to UTI /Risk factors for urinary tract infection

Incomplete bladder emptying :

- Bladder outflow obstruction
- Neurological problems (e.g. multiple sclerosis, diabetic neuropathy)
- Gynaecological abnormalities (e.g. uterine prolapse)
- Vesico-ureteric reflux

Foreign bodies :

- Urethral catheter or ureteric stent

Loss of host defences

- Atrophic urethritis and vaginitis in post menopausal women
- Diabetes mellitus

Clinical assessment

Typical features of cystitis and urethritis include:

- Abrupt onset of frequency of micturition and urgency
- Scalding pain in the urethra during micturition (dysuria)
- Suprapubic pain during and after voiding
- Intense desire to pass more urine after micturition, due to spasm of the inflamed bladder wall (strangury)
- Urine that may appear cloudy and have an unpleasant odour
- Microscopic or visible haematuria.

Systemic symptoms are usually slight or absent

1. However, infection in the lower urinary tract can spread; acute pyelonephritis is suggested by prominent systemic symptoms with fever, rigors, vomiting, hypotension and loin pain, guarding or tenderness, and may be an indication for hospitalisation.
2. Only about 30% of patients with acute pyelonephritis have associated symptoms of cystitis or urethritis.
3. Prostatitis is suggested by systemic symptoms and prostatic tenderness on rectal examination.

- The spectrum of presentations of urinary tract infection
  - Asymptomatic bacteriuria
- Symptomatic acute urethritis and cystitis
- Acute pyelonephritis
- Acute prostatitis
- Septicaemia (usually Gram-negative bacteria)

Differential diagnosis

1. The differential diagnosis of lower urinary tract symptoms includes urethritis due to sexually transmitted disease, notably chlamydia or Reiter's syndrome.
2. Some patients, usually female, have symptoms suggestive of urethritis and cystitis but no bacteria are cultured from the urine (the 'urethral syndrome').
3. Possible explanations include infection with organisms not readily cultured by ordinary methods (e.g. *Chlamydia,* certain anaerobes), intermittent or low-count bacteriuria, reaction to toiletries or disinfectants, symptoms related to sexual intercourse, or post-menopausal atrophic vaginitis.
4. The differential diagnosis of acute pyelonephritis includes acute appendicitis, diverticulitis, cholecystitis, salpingitis, ruptured ovarian cyst or ectopic pregnancy.
5. In perinephric abscess, there is marked pain and tenderness, and often bulging of the loin on the affected side.
6. Patients are extremely ill, with fever, leucocytosis and positive blood cultures, but urinary symptoms are absent and urine contains neither pus cells nor organisms.

Investigations urine

1. In an otherwise healthy woman with a single lower urinary tract infection, urine culture prior to treatment is not mandatory.
2. It is necessary, however, in patients with recurrent infection or after failure of initial treatment, during pregnancy, or in patients susceptible to serious infection (e.g. the immunocompromised, those with diabetes or an indwelling catheter, or older people
3. Definitive diagnosis rests on the combination of typical clinical features with findings in the urine.
4. Most urinary pathogens can reduce nitrate to nitrite, and neutrophils and nitrites can usually be detected in symptomatic infections by urine dipstick tests for leucocyte esterase and nitrite, respectively.
5. The absence of both nitrites and leucocyte esterase in the urine makes UTI unlikely. Interpretation of bacterial counts in the urine, and of what is a 'significant' culture result, is based on probabilities.
6. Urine taken by suprapubic aspiration should be sterile, so the presence of any organisms is significant.
7. If the patient has symptoms and there are neutrophils in the urine, a small number of organisms is significant.
8. In asymptomatic patients> 10^9 organisms/ml is usually regarded as significant (asymptomatic bacteriuria).
9. Typical organisms causing UTI in the community include *E. coli* derived from the gastrointestinal tract (about 75% of infections), *Proteus spp., Pseudomonas spp., streptococci* and *Staphylococcus epidermidis.*
10. In hospital, *E. coli* still predominates, but *Klebsiella* or streptococci are more common.
11. Certain strains of E. coli have a particular propensity to invade the urinary tract.

Investigations to detect underlying predisposing factors for UTI are used selectively, most commonly in patients with recurrent infections.

Investigation of patients with urinary tract infection

All patients :

- Dipstick* estimation of nitrite, leucocyte esterase and glucose
- Microscopy/cytometry of urine for white blood cells, organisms

- Urine culture

Infants, children, and anyone with fever or complicated infection :

- Full blood count; urea, electrolytes, creatinine

Blood cultures

Pyelonephritis; males; children; women with recurrent infections :

- Renal tract ultrasound or CT
- Pelvic examination in women, rectal examination in men

Continuing haematuria or other suspicion of bladder lesion :

- Cystoscopym

Prostatomegaly

Benign Prostatic Hyperplasia (BPH)

- Benign prostate enlargement (BPE) is the medical term to describe an enlarged prostate.
- Benign prostatic hyperplasia (BPH), also known as benign prostatic hypertrophy, is a histologic diagnosis characterized by proliferation of the cellular elements of the prostate, leading to an enlarged prostate gland.
- Chronic bladder outlet obstruction (BOO) secondary to BPH may lead to urinary retention, impaired kidney function, recurrent urinary tract infections, gross hematuria, and bladder calculi.
- Benign prostate enlargement (BPE)

Signs and symptoms

- Constriction for the flow of urine due to enlarged prostate
- Nerves within the prostate and bladder may also play a role in causing the following common symptoms:

Urinary frequency

Urinary urgency

Nocturia- Needing to get up frequently at night to urinate

Hesitancy - Difficulty initiating the urinary stream; interrupted, weak stream

Incomplete bladder emptying - The feeling of persistent residual urine, regardless of the frequency of urination

Straining - The need strain or push (Valsalva maneuver) to initiate and maintain urination in order to more fully empty the bladder

Decreased force of stream - The subjective loss of force of the urinary stream over time

Dribbling - The loss of small amounts of urine due to a poor urinary stream as well as weak urinary stream

Diagnosis

Digital rectal examination : The digital rectal examination (DRE) is an integral part of the evaluation in men with presumed BPH. With the DRE, the examiner can assess prostate size and contour, evaluate for nodules, and detect areas suggestive of malignancy.

Laboratory studies

Urinalysis - Examine the urine using dipstick methods and/or via centrifuged sediment evaluation to assess for the presence of blood, leukocytes, bacteria, protein, or glucose

Urine culture - This may be useful to exclude infectious causes of irritative voiding and is usually performed if the initial urinalysis findings indicate an abnormality

Prostate-specific antigen (PSA) - Although BPH does not cause prostate cancer, men at risk for BPH are also at risk for this disease and should be screened accordingly (although screening for prostate cancer remains controversial)

Electrolytes, blood urea nitrogen (BUN), and creatinine - These evaluations are useful screening tools for chronic kidney disease in patients who have high postvoid residual (PVR) urine volumes; however, a routine serum creatinine measurement is not indicated in the initial evaluation of men with lower urinary tract symptoms (LUTS) secondary to BPH.

Ultrasonography

Ultrasonography (abdominal, renal, transrectal) is useful for helping to determine bladder and prostate size and the degree of hydronephrosis (if any) in patients with urinary retention or signs of kidney insufficiency. Generally, it is not indicated for the initial evaluation of uncomplicated LUTS.

Endoscopy of the lower urinary tract

Cystoscopy may be indicated in patients scheduled for invasive treatment or in whom a foreign body or malignancy is suspected. In addition, endoscopy may be indicated in patients with a history of sexually transmitted disease (eg, gonococcal urethritis), prolonged catheterization, or trauma.

IPSS/AUA-SI

The severity of BPH can be determined with the International Prostate Symptom Score (IPSS)/ American Urological Association Symptom Index (AUA-SI) plus a disease-specific quality of life (QOL) question.

Other tests

Flow rate - Useful in the initial assessment and to help determine the patient's response to treatment

PVR urine volume - Used to gauge the severity of bladder decompensation; it can be obtained invasively with a catheter or noninvasively with a transabdominal ultrasonic scanner

Pressure-flow studies - Findings may prove useful for evaluating for BOO

Urodynamic studies - To help distinguish poor bladder contraction ability (detrusor underactivity) from BOO

Cytologic examination of the urine - May be considered in patients with predominantly irritative voiding symptoms

Nephrotic Syndrome

Nephrotic syndrome describes the onset of heavy proteinuria (>3.0 g/24 h), hypertension, hypercholesterolemia, hypoalbuminemia, edema/ anasarca, and microscopic hematuria; if only large amounts of proteinuria are present without clinical manifestations, the condition is sometimes called nephrotic-range proteinuria. The glomerular filtration rate (GFR) in these patients may initially be normal or, rarely, higher than normal, but with persistent hyperfiltration and continued nephron loss, it typically declines over months to years. Patients with a basement membrane syndrome either have genetically abnormal basement membranes (Alport's syndrome) or an autoimmune response to basement membrane collagen IV (Goodpasture's syndrome) associated with microscopic hematuria, mild to heavy proteinuria, and hypertension with variable elevations in serum creatinine. Glomerular-vascular syndrome describes patients with vascular injury producing hematuria and moderate proteinuria. Affected individuals can have vasculitis, thrombotic microangiopathy, antiphospholipid syndrome, or, more commonly, a systemic disease such as atherosclerosis, cholesterol emboli, hypertension, sickle cell anemia, and autoimmunity. Infectious disease-associated syndrome is most important if one has an international perspective. Save for subacute bacterial endocarditis in the Western Hemisphere, malaria and schistosomiasis may be the most common causes of glomerulonephritis throughout the world, closely followed by HIV and chronic hepatitis B and C. These infectious diseases produce a variety of inflammatory reactions in glomerular capillaries, ranging from nephrotic syndrome to acute nephritic injury, and urinalyses that demonstrate a combination of hematuria and proteinuria.

Nephrotic syndrome is kidney disease with proteinuria, hypoalbuminemia, and edema. Nephrotic-range proteinuria is 3 grams per day or more. On a single spot urine collection, it is 2 g of protein per gram of urine creatinine.

Nephrotic syndrome may affect adults and children, of both sexes and of any race. It may occur in typical form, or in association with nephritic syndrome. The latter connotes glomerular inflammation, with hematuria and impaired kidney function.

Classification

Nephrotic syndrome can be primary; being a disease specific to the kidneys, or it can be secondary, being a renal manifestation of a systemic general illness. In all cases, injury to glomeruli is an essential feature.

Primary causes of nephrotic syndrome include the following, in approximate order of frequency:

- Minimal-change nephropathy
- Focal glomerulosclerosis
- Membranous nephropathy
- Hereditary nephropathies

Secondary causes include the following, again in order of approximate frequency:

- Diabetes mellitus
- Lupus erythematosus
- Amyloidosis and paraproteinemias
- Viral infections (eg, hepatitis B, hepatitis C, human immunodeficiency virus [HIV])
- Preeclampsia

Nephrotic-range proteinuria may occur in other kidney diseases, such as IgA nephropathy. In that common glomerular disease, one third of patients may have nephrotic-range proteinuria.

Nephrotic syndrome may occur in persons with sickle cell disease and evolve to renal failure. Membranous nephropathy may complicate bone marrow transplantation, in association with graft versus host disease.

Kidney diseases that affect tubules and interstitium, such as interstitial nephritis, will not cause nephrotic syndrome.

Nephrotic syndrome classically presents with heavy proteinuria, minimal hematuria, hypoalbuminemia, hypercholesterolemia, edema, and hypertension. If left undiagnosed or untreated, some of these syndromes will progressively damage enough glomeruli to cause a fall in GFR, producing renal failure.

Clinical Features

The first sign of nephrotic syndrome in children is usually swelling of the face; this is followed by swelling of the entire body. Adults can present with dependent edema. Foamy urine may be a presenting feature. A thrombotic complication, such as deep venous thrombosis of the calf veins or even a pulmonary embolus, may be the first clue indicating nephrotic syndrome.

Additional historical features can be related to the cause of nephrotic syndrome. Thus, the recent start of a nonsteroidal anti-inflammatory drug (NSAID) or a 10-year history of diabetes may be very relevant.

Difference between acute nephritis and nephrotic syndrome

Acute nephritis	Nephrotic syndrome
• Characterized by hematuria, edema, hypertension, oligouria	• Characterized by heavy proteinuria, hpo album-inemia, edema, hperli-pidemia
• 90% post infective immune complex	• 90% idiopathic
• Usually only 1 attack	• Relapses common
• Immune complex deposition	• Retraction of epithelial foot process
• Urine: Alb 1+/2+, hematuria, RBC cast	• Urine: Selective proteinuria, No RBC
• Blood urea/creat raised	• Blood urea/ creat normal

Differentiation Between Nephrotic Syndrome and Nephritic Syndrome

Onset	Insidious	Abrupt
Edema	++	++++
Blood pressure	Normal	Raised
Jugular venous pressure	Normal/low	Raised
Proteinuria	++	++++
Nematuria	May/may not occur	+++
Red blood cell casts	Absent	Present
Serum albumin	Low	Normal/slightly reduced

Diagnostic Criteria-Nephritic Syndrome

- Inflammatory rapture of glomerular capillaries resulting in bleeding into urinary space

(Bowman's capsule). Protefnuria and oedema mild.

- Oliguria - Reduced GFR causing reduced urine output.
- Azotemia - elevated BUN.
- Hypertension - increased fluid retention & Renin-Angiotension-Aldosterone activation by ischaemic kindneys
- Hematuria - form RBC casts &: granular casts

Diagnostic Criteria for Nephrotic Syndrome

Factor	Criteria
Heavy proteinuria	Spot urine showing a protein-to-creatinine ratio of > 3 to 3.5 mg protein/mg creatinine (300 to 350 mg/mmol), or 24-hour urine collection showing > 3 to 3.5g protin
Hypoalbuminemia	Serum albumin < 2.5 g per dL (25 g per L)
Edema	Clinical evidence of peripheral edema
Hyperlipidemia (not required	Severe hyperlipidemia, total cholesterol is often> 350 mg per DL (9.06 mmol per L)

Physical Examination

Edema is the predominant feature of nephrotic syndrome and initially develops around the eyes and legs. With time, the edema becomes generalized and may be associated with an increase in weight, the development of ascites, or pleural effusions. Hematuria and hypertension manifest in a minority of patients.

Additional features on examination will vary according to cause and as a result of whether or not renal function impairment exists. Thus, in the case of longstanding diabetes, there may be diabetic retinopathy, which correlates closely with diabetic nephropathy. If the kidney function is reduced, there may be hypertension and/or anemia.

Ancillary Investigations

Diagnostic studies for nephrotic syndrome may include the following:

- Urinalysis
- Urine sediment examination
- Urinary protein measurement
- Serum albumin
- Serologic studies for infection and immune abnormalities
- Renal ultrasonography
- Renal biopsy

Urine Studies

Urinalysis : Urinalysis is the first test used in the diagnosis of nephrotic syndrome. Nephrotic range proteinuria will be apparent by 3+ or 4+ readings on the dipstick, or by semiquantitative testing by sulfosalicylic acid. A 3+ reading represents 300 mg/dL of urinary protein or more, which is 3 g/L or more and thus in the nephrotic range.

Nephritis

Nephritis is a condition in which the nephrons, the functional units of the kidneys, become inflamed. This inflammation, which is also known as glomerulonephritis, can adversely affect kidney function.

Causes

Nephritis is often caused by infections, and toxins. In some cases, the cause may not be clear.

Nephritis and kidney disease often seem to run in families, which suggests a possible genetic component. Some infections, such as HIV and hepatitis B or C, can also cause nephritis.

In some cases, kidney damage can occur as a result of medications, such as antibiotics. This damage can lead to nephritis. Taking too many pain relievers, nonsteroidal anti-inflammatory drugs (NSAIDs), or diuretic pills can also cause this condition. Pyelonephritis is inflammation that results from a urinary tract infection that reaches the renal pelvis of the kidney.

Lupus nephritis is inflammation of the kidney caused by systemic lupus erythematosus (SLE), a disease of the immune system.

Athletic nephritis is nephritis resulting from strenuous exercise. Bloody urine after strenuous exercise may also result from March hemoglobinuria, which is caused by trauma to red blood cells, causing their rupture, which leads to the release of hemoglobin into the urine.

Risk factors : The most important risk factors for kidney disease are:

- A family history of kidney disease
- High blood pressure
- Diabetes
- Obesity
- Heart disease
- An age of 60 years or more

Types of Nephritis

There are several different types of nephritis, including:

Acute glomerulonephritis: This form of nephritis can develop suddenly after a severe infection, such as strep throat, hepatitis, or HIV. Lupus and rarer disorders, such as vasculitides and granulomatosis with polyangiitis (GPA), can also lead to acute inflammation of the kidneys. A person with these conditions will require prompt medical attention during a flare-up to reduce kidney damage.

Lupus nephritis: Lupus is an autoimmune disease, which means that the immune system mistakenly attacks healthy tissues in the body. Over half of all individuals with a lupus diagnosis eventually develop lupus nephritis. This occurs when the immune system attacks the kidneys.

Alport syndrome, or hereditary nephritis: This disease can lead to kidney failure, as well as vision and hearing problems. Alport syndrome is passed on in the genes, and it is usually more severe in men.

Chronic glomerulonephritis: This form of nephritis develops slowly and causes few symptoms in its early stages. As with acute glomerulonephritis, this condition can cause severe kidney damage and kidney failure. It may run in families or develop after a sudden disease.

IgA nephropathy: This is one of the more common forms of nephritis. It develops when IgA antibody deposits build up in the kidneys and cause inflammation.The immune system develops antibodies to combat harmful substances and organisms that enter the body. People with IgA neuropathy have defective IgA antibodies.

Interstitial nephritis: Often developing very rapidly, this form of nephritis usually occurs due to infection or a particular medication. It affects the part of the kidney called the interstitium, which is a fluid-filled space.

General symptoms

The symptoms of nephritis are rarely severe in the early stages. The following signs may indicate that a person has this condition:

- Changes in urinating habits
- Swelling anywhere in the body, especially the hands, feet, ankles, and face
- Changes in urine color
- Foamy urine
- Blood in the urine

Lupus nephritis

Lupus nephritis is histologically evident in most patients with systemic lupus erythematosus (SLE), even those without clinical manifestations of renal disease. Evaluating renal function in SLE patients is important because early detection and treatment of renal involvement can significantly improve renal outcome.

Aetiology

Genetic factors: As with many autoimmune disorders, evidence suggests that genetic predisposition plays an important role in the development of both SLE and lupus nephritis. Multiple genes, many of which are not yet identified, mediate this genetic predisposition.

Signs and symptoms

Patients with lupus nephritis may report the following :

- Other symptoms of active SLE (eg, fatigue, fever, rash, arthritis, serositis, or central nervous system [CNS] disease); these are more common with focal proliferative and diffuse proliferative lupus nephritis

- Asymptomatic lupus nephritis - During regular follow-up, laboratory abnormalities suggest active lupus nephritis; this is more typical of mesangial or membranous lupus nephritis.
- Active nephritis - Peripheral edema secondary to hypertension or hypoalbuminemia; extreme peripheral edema is more common with diffuse or membranous lupus nephritis.
- Diffuse lupus nephritis - Headache, dizziness, visual disturbances, or signs of cardiac decompensation

Physical findings may include the following:

- Focal and diffuse lupus nephritis - Generalized active SLE with the presence of a rash, oral or nasal ulcers, synovitis, or serositis; signs of active nephritis
- Active lupus nephritis - Hypertension, peripheral edema, and, occasionally, cardiac decompensation.
- Membranous lupus nephritis - Peripheral edema, ascites, and pleural and pericardial effusions without hypertension

Required Investigations

Laboratory tests to evaluate renal function in SLE patients include the following :

- Blood urea nitrogen (BUN) testing
- Serum creatinine assessment
- Urinalysis (to check for protein, red blood cells [RBCs], and cellular casts)
- Spot urine test for creatinine and protein concentration
- 24-hour urine test for creatinine clearance and protein excretion

Laboratory tests for SLE disease activity include the following:

- Antibodies to double-stranded DNA (dsDNA)
- Complement (C3, C4, and *CH50*)
- Erythrocyte sedimentation rate (ESR)
- C-reactive protein (CRP)

Renal biopsy should be considered in any patient with SLE who has clinical or laboratory evidence of active nephritis, especially upon the first episode of nephritis.

Lupus nephritis is staged according to the classification revised by the International Society of Nephrology (ISN) and the Renal Pathology Society (RPS) in 2003, as follows :

- Class I - Minimal mesangial lupus nephritis
- Class II - Mesangial proliferative lupus nephritis
- Class III - Focal lupus nephritis (active and chronic; proliferative and sclerosing)
- Class IV - Diffuse lupus nephritis (active and chronic; proliferative and sclerosing; segmental and global)
- Class V - Membranous lupus nephritis
- Class VI - Advanced sclerosis lupus nephritis

Tubulointerstitial Nephritis

The term tubulointerstitial is used to broadly refer to kidney diseases that involve structures in the kidney outside the glomerulus. These diseases generally involve tubules and/or the interstitium of the kidney and spare the glomeruli.

Kidney biopsy : This is an example of acute interstitial nephritis. The renal cortex shows a diffuse interstitial, predominantly mononuclear, inflammatory infiltrate with no changes to the glomerulus. Tubules in the center of the field are separated by inflammation and edema, as compared with the more normal architecture in the right lower area (periodic acid-Schiff, 40 X).

Although primary glomerular diseases are often associated with prominent tubulointerstitial changes, the clinical presentation is dominated by the consequences of glomerular injury.

Kidney biopsy in interstitial nephritis.Acute crescentic glomerulonephritis. The glomerular tuft is compressed by the proliferation of epithelial cells, forming a crescent. The interstitium shows mononuclear inflammatory infiltrate and edema (periodic acid-Schiff, 40 X).

Aetiology

Tubulointerstitial diseases of the kidney encompass diverse etiologies and pathophysiologic processes, and the patient can present with acute or chronic conditions. Many forms of tubulointerstitial injury involve exposure to drugs or other nephrotoxic agents such as heavy metals and, rarely, infection. By far the most common form of tubulointerstitial inflammation is hypersensitivity reaction to medications, termed allergic interstitial nephritis.

History and Physical Examination

The history of patients with tubulointerstitial nephritis depends on whether the disease is acute or chronic. A thorough physical examination may provide clues to the diagnosis (eg, fever, rash in acute tubulointerstitial nephritis, lividoreticularis and Hollenhorst plaques in the optic fundi in atheroembolic disease), but, in most patients, no characteristic findings exist. Some patients present with hypertension, although others may be normotensive or hypotensive (eg, Balkan endemic nephropathy).

Investigations

CBC With Differential: Eosinophilia, when present, can be very helpful in the evaluation of tubulointerstitial nephritis. Typically, eosinophilia is absent in acute tubulointerstitial nephritis that is induced by nonsteroidal anti-inflammatory drugs (NSAIDs).

Chemistry Panel

A complete set of chemistries, including blood urea nitrogen (BUN) and serum creatinine, provides information on whether renal insufficiency exists. A low bicarbonate level (total carbon dioxide < 24-23 mEq/L) may indicate acidosis. Low serum potassium levels may indicate a proximal tubular disorder, and elevated serum potassium levels with a low bicarbonate level may indicate type 4 renal tubular acidosis, which can be observed with lead nephropathy and nonsteroidal anti-inflammatory drug (NSAID)-induced analgesic nephropathy, among other conditions.

Urine Studies

Urinalysis may reveal proteinuria, hematuria, and the presence of white blood cells (WBCs), with or without bacteria. A microscopic analysis of urine sediment may reveal casts, WBCs, eosinophils, and crystals. If allergic interstitial nephritis is suspected, send a cytospin specimen to determine if eosinophils are in the urine. Unfortunately, the absence of eosinophiluria does not rule out the diagnosis, and it can be observed in other diseases, including cholesterol microembolism, urinary tract infections, parasitic disorders, and glomerulonephritis. In nonsteroidal anti-inflammatory drug (NSAID)-induced acute tubulointerstitial nephritis, eosinophiluria is usually absent.

Ultrasonography and Radiography

Ultrasonography is noninvasive imaging technique that is extremely helpful in identifying hydronephrosis in obstructive disease as well as calculi in stone disease. Both radiolucent and radiopaque stones can be visualized with this modality. A combination of ultrasonography and flat plate kidney, ureter, and bladder (KUB) radiography is helpful in the workup and identification of radiopaque versus radiolucent stones.

Kidney Biopsy and Histologic Features

Kidney biopsy is the definitive test for diagnosing acute allergic interstitial nephritis, particularly in cases in which the clinical diagnosis is difficult. Because the differential diagnosis of acute tubulointerstitial nephritis encompasses multiple etiologies, consider kidney biopsy when the diagnosis is not obvious.

Kidney biopsy shows mononuclear and often eosinophilic cellular infiltration of the renal parenchyma with sparing of the glomeruli. Sometimes, interstitial changes such as fibrosis and atrophy are also present (eg, Renal biopsies have shown severe interstitial fibrosis in patients with Chinese herb/aristolochic acid nephropathy).

Acute Kideny Injury

Acute kidney injury (AKI), previously known as acute renal failure, is characterized by the sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI is not a single disease but, rather, a designation for a heterogeneous group of conditions that share common diagnostic features: specifically, an increase in the blood urea nitrogen (BUN) concentration and/or an increase in the plasma or serum creatinine (SCr) concentration often associated with a reduction in urine volume. AKI can range in severity from asymptomatic and transient changes in laboratory parameters of glomerular filtration rate (GFR), to overwhelming and rapidly fatal derangements in effective circulating volume regulation and electrolyte and acid-base composition of the plasma.

Changing the name of a syndrome as well known as "acute renal failure" does not occur frequently. We will summarize some of the reasons why the name was changed to "acute kidney injury." The term failure reflects only part of the spectrum of damage to the kidney that occurs clinically. In most cases of damage, the reduction in kidney function is modest. Nevertheless, this modest change has been documented to be associated with negative effects on outcome, albeit not nearly as ominous as seen with large decreases in kidney function associated with frank kidney failure that often requires acute dialysis therapies. Furthermore, the term renal is not well understood in the general population and this makes communication with patients and family more challenging; hence "kidney" has replaced "renal."

Categories of AKI

AKI may be classified into 3 general categories, as follows:

- Prerenal - As an adaptive response to severe volume depletion and hypotension, with structurally intact nephrons
- Intrinsic - In response to cytotoxic, ischemic, or inflammatory insults to the kidney, with structural and functional damage
- Postrenal - From obstruction to the passage of urine

Etiology

The etiology of AKI consists of 3 main mechanisms: prerenal, intrinsic, and obstructive.

Prerenal AKI

Prerenal AKI represents the most common form of kidney injury and often leads to intrinsic AKI if it is not promptly corrected. Volume loss can provoke this syndrome.

Volume depletion can be caused by the following :

- Renal losses - Diuretics, polyuria
- GI losses - Vomiting, diarrhea
- Cutaneous losses - Burns, Stevens-Johnson syndrome
- Hemorrhage
- Pancreatitis

Decreased cardiac output can be caused by the following:

- Heart failure
- Pulmonary embolus
- Acute myocardial infarction
- Severe valvular disease
- Abdominal compartment syndrome - Tense ascites

Systemic vasodilation can be caused by the following:

- Sepsis
- Anaphylaxis
- Anesthetics
- Drug overdose

Afferent arteriolar vasoconstriction can be caused by the following:

- Hypercalcemia
- Drugs - NSAIDs, amphotericin B, calcineurin inhibitors, norepinephrine, radiocontrast agents
- Hepatorenal syndrome

Diseases that decrease effective arterial blood volume include the following:

- Hypovolemia
- Heart failure
- Liver failure
- Sepsis

Renal arterial diseases that can result in AKI include renal arterial stenosis, especially in the setting of hypotension or initiation of ACE inhibitors or ARBs. Renal artery stenosis typically results from atherosclerosis or fibromuscular dysplasia, but is also a feature of the genetic syndromes type 1 neurofibromatosis, Williams syndrome, and Alagille syndrome.

Patients can also develop septic embolic disease (eg, from endocarditis) or cholesterol emboli, often as a result of instrumentation or cardiovascular surgery.

Intrinsic AKI

Structural injury in the kidney is the hallmark of intrinsic AKI.

Vascular (large- and small-vessel) causes of intrinsic AKI include the following :

- Renal artery obstruction - Thrombosis, emboli, dissection, vasculitis
- Renal vein obstruction - Thrombosis
- Microangiopathy - Thrombotic thrombocytopenic purpura (TTP), Hemolytic-uremic syndrome (HUS), disseminated intravascular coagulation (DIC), preeclampsia
- Malignant hypertension
- Scleroderma renal crisis
- Transplant rejection
- Atheroembolic disease

Glomerular causes include the following:

- Anti-glomerular basement membrane (GBM) disease - As part of Goodpasture syndrome or renal limited disease
- Anti-neutrophil cytoplasmic antibody-associated glomerulonephritis (ANCA-associated glomerulone-phritis) - Wegener granulomatosis, Churg-Strauss syndrome, microscopic polyangiitis
- Immune complex glomerulonephritis - Lupus, postinfectious glomerulonephritis, cryoglobulinemia, primary membranoproliferative glomerulonephritis

Tubular etiologies may include ischemia or cytotoxicity. Cytotoxic etiologies include the following:

- Heme pigment - Rhabdomyolysis, intravascular hemolysis
- Crystals - Tumor lysis syndrome, seizures, ethylene glycol poisoning, megadose vitamin C, acyclovir, indinavir, methotrexate
- Drugs - Aminoglycosides, lithium, amphotericin B, pentamidine, cisplatin, ifosfamide, radiocontrast agents

Interstitial causes include the following:

- Drugs - Penicillins, cephalosporins, NSAIDs, proton-pump inhibitors, allopurinol, rifampin, indinavir, mesalamine, sulfonamides
- Infection - Pyelonephritis, viral nephritides
- Systemic disease - Surgren syndrome, sarcoid, lupus, lymphoma, leukemia, tubulonephritis, uveitis

Postrenal AKI

Mechanical obstruction of the urinary collecting system, including the renal pelvis, ureters, bladder, or urethra, results in obstructive uropathy or postrenal AKI.

Causes of obstruction include the following:

Causes of postrenal AKI include the following:

- Ureteric obstruction - Stone disease, tumor, fibrosis, ligation during pelvic surgery
- Bladder neck obstruction - Benign prostatic hypertrophy (BPH), cancer of the prostate (CA prostate or prostatic CA), neurogenic bladder, tricyclic antidepressants, ganglion blockers, bladder tumor, stone disease, hemorrhage/clot
- Urethral obstruction - Strictures, tumor, phimosis
- Intra-abdominal hypertension - Tense ascites
- Renal vein thrombosis

Diseases causing urinary obstruction from the level of the renal tubules to the urethra include the following:

- Tubular obstruction from crystals - Eg, uric acid, calcium oxalate, acyclovir, sulfonamide, methotrexate, myeloma light chains

- Ureteral obstruction - Retroperitoneal tumor, retroperitoneal fibrosis (methysergide, propranolol, hydralazine), urolithiasis, or papillary necrosis
- Urethral obstruction - Benign prostatic hypertrophy; prostate, cervical, bladder, or colorectal carcinoma; bladder hematoma; bladder stone; obstructed Foley catheter; neurogenic bladder; stricture

Etiology in newborns and infants

Prerenal AKI

The patient's age has significant implications for the differential diagnosis of AKI. In newborns and infants, causes of prerenal AKI include the following:

- Perinatal hemorrhage - Twin-twin transfusion, complications of amniocentesis, abruptio placenta, birth trauma
- Neonatal hemorrhage - Severe intraventricular hemorrhage, adrenal hemorrhage
- Perinatal asphyxia and hyaline membrane disease (newborn respiratory distress syndrome) - Both may result in preferential blood shunting away from the kidneys (ie, prerenal) to central circulation

Intrinsic AKI

Causes of intrinsic AKI include the following:

- ATN - Can occur in the setting of perinatal asphyxia; ATN also has been observed secondary to medications (eg, aminoglycosides, NSAIDs) given to the mother perinatally
- ACE inhibitors - Can traverse the placenta, resulting in a hemodynamically mediated form of AKI
- Acute glomerulonephritis – Rare; most commonly the result of maternal-fetal transfer of antibodies against the neonate's glomeruli or transfer of chronic infections (syphilis, cytomegalovirus) associated with acute glomerulonephritis

Postrenal AKI

Congenital malformations of the urinary collecting systems should be suspected in cases of postrenal AKI.

Pathophysiology

The driving force for glomerular filtration is the pressure gradient from the glomerulus to the Bowman space. Glomerular pressure depends primarily on renal blood flow (RBF) and is controlled by the combined resistances of renal afferent and efferent arterioles. Regardless of the cause of AKI, reductions in RBF represent a common pathologic pathway for decreasing glomerular filtration rate (GFR).

Clinical Features

Prerenal azotemia should be suspected in the setting of vomiting, diarrhea, glycosuria causing polyuria, and several medications including diuretics, NSAIDs, ACE inhibitors, and ARBs.

Physical Examination

Physical signs of orthostatic hypotension, tachycardia, reduced jugular venous pressure, decreased skin turgor, and dry mucous membranes are often present in prerenal azotemia.

A history of prostatic disease, nephrolithiasis, or pelvic or paraaortic malignancy would suggest the possibility of postrenal AKI. Whether or not symptoms are present early during obstruction of the urinary tract depends on the location of obstruction. Colicky flank pain radiating to the groin suggests acute ureteric obstruction. Nocturia and urinary frequency or hesitancy can be seen in prostatic disease. Abdominal fullness and suprapubic pain can accompany massive bladder enlargement. Definitive diagnosis of obstruction requires radiologic investigations.

A careful review of all medications is imperative in the evaluation of an individual with AKI. Not only are medications frequently a cause of AKI but doses of administered medications must be adjusted for estimated GFR. Idiosyncratic reactions to a wide variety of medications can lead to allergic interstitial nephritis, which may be accompanied by fever, arthralgias, and a pruritic erythematous rash. The absence of systemic features of hypersensitivity, however, does not exclude the diagnosis of interstitial nephritis.

AKI accompanied by palpable purpura, pulmonary hemorrhage, or sinusitis raises the possibility of systemic vasculitis with glomerulonephritis. Atheroembolic disease can be associated with livedo reticularis and other signs of emboli to the legs. A tense abdomen should prompt consideration of acute abdominal compartment syndrome, which requires measurement of bladder pressure. Signs of limb ischemia may be clues to the diagnosis of rhabdomyolysis.

Urine Findings

Complete anuria early in the course of AKI is uncommon except in the following situations: complete urinary tract obstruction, renal artery occlusion, overwhelming septic shock, severe ischemia (often with cortical necrosis), or severe proliferative glomerulonephritis or vasculitis.

A reduction in urine output (oliguria, defined as <400 mL/24 h) usually denotes more significant AKI (i.e., lower GFR) than when urine output is preserved.

Oliguria is associated with worse clinical outcomes. Preserved urine output can be seen in nephrogenic diabetes insipidus characteristic of long standing urinary tract obstruction, tubulointerstitial disease, or nephrotoxicity from cisplatin or aminoglycosides, among other causes. Red or brown urine may be seen with or without gross hematuria; if the color persists in the supernatant after centrifugation, then pigment nephropathy from rhabdomyolysis or hemolysis should be suspected.

The urinalysis and urine sediment examination are invaluable tools, but they require clinical correlation because of generally limited sensitivity and specificity. In the absence of preexisting proteinuria from CKD, AKI from ischemia or nephrotoxins leads to mild proteinuria (<1 g/d). Greater proteinuria in AKI suggests damage to the glomerular ultrafiltration barrier or excretion of myeloma light chains; the latter are not detected with conventional urine dipsticks (which detect albumin) and require the sulfosalicylic acid test or immunoelectrophoresis. Atheroemboli can cause a variable degree of proteinuria. Extremely heavy proteinuria ("nephrotic range," >3.5 g/d) can occasionally be seen in glomerulonephritis, vasculitis, or interstitial nephritis (particularly from NSAIDs). AKI can also complicate cases of minimal change disease, a cause of the nephrotic syndrome. If the dipstick is positive for hemoglobin but few red blood cells are evident in the urine sediment, then rhabdomyolysis or hemolysis should be suspected.

Physical Examination

Obtaining a thorough physical examination is extremely important when collecting evidence about the etiology of AKI. Clues may be found in any of the following: Skin; Eyes; Ears; Cardiovascular system; Abdomen and Pulmonary system.

Skin

Skin examination may reveal the following:

- Livido reticularis, digital ischemia, butterfly rash, palpable purpura - Systemic vasculitis
- Maculopapular rash - Allergic interstitial nephritis
- Track marks (ie, intravenous drug abuse) - Endocarditis

Petechiae, purpura, ecchymosis, and livedo reticularis provide clues to inflammatory and vascular causes of AK. Infectious diseases, thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation (DIC), and embolic phenomena can produce typical cutaneous changes.

Eyes and ears

Eye examination may reveal the following:

- Keratitis, iritis, uveitis, dry conjunctivae - Autoimmune vasculitis
- Jaundice - Liver diseases
- Band keratopathy (ie, hypercalcemia) - Multiple myeloma

- Signs of diabetes mellitus
- Signs of hypertension
- Atheroemboli - Retinopathy

Evidence of uveitis may indicate interstitial nephritis and necrotizing vasculitis. Ocular palsy may indicate ethylene glycol poisoning or necrotizing vasculitis. Findings suggestive of severe hypertension, atheroembolic disease, and endocarditis may be observed on careful examination of the eyes.

Ear examination may reveal the following:

- Hearing loss - Alport disease and aminoglycoside toxicity
- Mucosal or cartilaginous ulcerations - Wegener granulomatosis

Cardiovascular system

The most important part of the physical examination is the assessment of cardiovascular and volume status. The physical examination must include the following:

- Pulse rate and blood pressure recordings measured in the supine and the standing position
- Close inspection of the jugulovenous pulse
- Careful examination of the heart and lungs, skin turgor, and mucous membranes
- Assessment for peripheral edema

Cardiovascular examination may reveal the following:

- Irregular rhythms (ie, atrial fibrillation) - Thromboemboli
- Murmurs - Endocarditis
- Pericardial friction rub - Uremic pericarditis
- Increased jugulovenous distention, rales, S_3- Heart failure

In hospitalized patients, accurate daily records of fluid intake and urine output, as well as daily measurements of patient weight, are important. Hypovolemia leads to hypotension; however, hypotension may not necessarily indicate hypovolemia.

Severe heart failure may also cause hypotension. Although patients with heart failure may have low blood pressure, volume expansion is present and effective renal perfusion is poor, which can result in AKI.

Severe hypertension with renal failure suggests one of the following disorders:

- Renovascular disease
- Glomerulonephritis
- Vasculitis
- Atheroembolic disease

Abdomen

Abdominal examination may reveal the following:

- Pulsatile mass or bruit - Atheroemboli
- Abdominal or costovertebral angle tenderness - Nephrolithiasis, papillary necrosis, renal artery thrombosis, renal vein thrombosis
- Pelvic, rectal masses; prostatic hypertrophy; distended bladder – Urinary obstruction
- Limb ischemia, edema - Rhabdomyolysis

Abdominal examination findings can be useful in helping to detect obstruction at the bladder outlet as the cause of renal failure; such obstruction may be due to cancer or to an enlarged prostate.

The presence of tense ascites can indicate elevated intra-abdominal pressure that can retard renal venous return and result in AKI. The presence of an epigastric bruit suggests renal vascular hypertension, which may predispose to AKI.

Pulmonary system

Pulmonary examination may reveal the following :

- Rales - Goodpasture syndrome, Wegener granulomatosis
- Hemoptysis - Wegener granulomatosis

Ancillary Investigations

Several laboratory tests, including the following, are useful for assessing the etiology of acute kidney injury (AKI) and can aid in proper management of the disease:

- Complete blood count (CBC)
- Serum biochemistries

- Urine analysis with microscopy
- Urine electrolytes

In some cases, renal imaging is useful, especially if renal failure is secondary to obstruction. The American College of Radiology recommends ultrasonography, preferably with Doppler methods, as the most appropriate imaging method in AKI.

Kidney Function Studies

Although increased levels of blood urea nitrogen (BUN) and creatinine are the hallmarks of renal failure, the rate of rise depends on the degree of renal insult and, with respect to BUN, on protein intake. BUN may be elevated in patients with gastrointestinal (GI) or mucosal bleeding, steroid treatment, or protein loading.

The ratio of BUN to creatinine is an important finding. The ratio can exceed 20:1 in conditions in which enhanced reabsorption of urea is favored (eg, in volume contraction); this suggests prerenal AKI.

Urinalysis

Findings of granular, muddy brown casts are highly suggestive of tubular necrosis. The presence of tubular cells or tubular cell casts also supports the diagnosis of ATN. Often, oxalate crystals are observed in cases of ATN.

Fractional Excretion of Sodium and Urea

Sodium : Urine electrolyte findings also can serve as valuable indicators of functioning renal tubules. The fractional excretion of sodium (FENa) is the commonly used indicator. However, the interpretation of results from patients in nonoliguric states, those with glomerulonephritis, and those receiving or ingesting diuretics can lead to an erroneous diagnosis.

FENa can be a valuable test for helping to detect extreme renal avidity for sodium in conditions such as hepatorenal syndrome.

Urea

In patients who are receiving diuretics, a fractional excretion of urea (FEUrea) can be obtained, since urea transport is not affected by diuretics.

Bladder Pressure

An intra-abdominal pressure of less than 10 mm Hg is considered normal and suggests that abdominal compartment syndrome is not the cause of AKI. An intra-abdominal pressure above 10 mm Hg is abnormal, but patients who have pressures of 15-25 mm Hg are at particular risk for abdominal compartment syndrome, and those with bladder pressures above 25 mm Hg should be suspected of having AKI as a result of abdominal compartment syndrome.

Emerging Biomarkers

Creatinine elevation is a late marker for renal dysfunction and, once elevated, reflects a severe reduction in glomerular filtration rate (GFR). Consequently, a number of biomarkers are being investigated to risk stratify and predict AKI in patients at risk for the disease.

Ultrasonography

Renal ultrasonography is useful for evaluating existing renal disease and obstruction of the urinary collecting system. Obtaining images of the kidneys can be technically difficult in patients who are obese, however, as well as in those with abdominal distention from ascites, gas, or retroperitoneal fluid collection.

Doppler ultrasonography

Doppler scans are useful for detecting the presence and nature of renal blood flow. Because renal blood flow is reduced in prerenal and intrarenal AKI, findings are of little use in the diagnosis of AKI.

Nuclear Scanning

Radionuclide imaging with technetium-99m-mercaptoacetyltriglycine (^{99m} Tc-MAG3), ^{99m} Tc-diethylene-triamine penta-acetic acid (^{99m} Tc-DTPA), or iodine-131 (^{131} I)-hippurate can be used to assess renal blood flow, as well as tubular function.

Aortorenal Angiography

Aortorenal angiography can be helpful in establishing the diagnosis of renal vascular diseases, including the following:

- Renal artery stenosis
- Renal atheroembolic disease
- Atherosclerosis with aortorenal occlusion
- Certain cases of necrotizing vasculitis (eg, polyarteritis nodosa)

Renal Biopsy

A renal biopsy may also be indicated when renal function does not return for a prolonged period and a prognosis is required to develop long-term management. In as many as 40% of cases, renal biopsy results reveal an unexpected diagnosis.

Chronic Kidney Disease

Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR). The term chronic renal failure applies to the process of continuing significant irreversible reduction in nephron number and typically corresponds to CKD stages 3–5. The pathophysiologic processes and adaptations associated with chronic renal failure. The dispiriting term end-stage renal disease represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the uremic syndrome. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation.

Pathophysiology of Chronic Kidney Disease

The pathophysiology of CKD involves two broad sets of mechanisms of damage: (1) initiating mechanisms specific to the underlying etiology (e.g., genetically determined abnormalities in kidney development or integrity, immune complex deposition and inflammation in certain types of glomerulonephritis, or toxin exposure in certain diseases of the renal tubules and interstitium) and (2) a set of progressive mechanisms, involving hyperfiltration and hypertrophy of the remaining viable nephrons, that are a common consequence following long-term reduction of renal mass, irrespective of underlying etiology. The responses to reduction in nephron number are mediated by vasoactive hormones, cytokines, and growth factors. Eventually, these short-term adaptations of hypertrophy and hyperfiltration become maladaptive as the increased pressure and flow predisposes to distortion of glomerular architecture, associated with sclerosis and dropout of the remaining nephrons. Increased intrarenal activity of the renin-angiotensin axis appears to contribute both to the initial adaptive hyperfiltration and to the subsequent maladaptive hypertrophy and sclerosis, the latter, in part, owing to the stimulation of transforming growth factor (TGF-).

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of the renin-angiotensin axis appears to contribute both to the initial adaptive hyperfiltration and to the subsequent maladaptive hypertrophy and sclerosis, the latter, in part, owing to the stimulation of transforming growth factor (TGF-). This process explains why a reduction in renal mass from an isolated insult may lead to a progressive decline in renal function over many years.

Identification of Risk Factors and Staging of CKD.

It is important to identify factors that increase the risk for CKD, even in individuals with normal GFR. Risk factors include hypertension, diabetes mellitus, autoimmune disease, older age, African ancestry, a family history of renal disease, a previous episode of acute kidney injury, and the presence of proteinuria, abnormal urinary sediment, or structural abnormalities of the urinary tract. Recent research in the genetics of predisposition to common complex diseases has revealed DNA sequence variants at a number of genetic loci that are associated with common forms of CKD. In order to stage CKD, it is necessary to estimate the GFR.

Pathophysiology and Biochemistry of Uremia

Although serum urea and creatinine concentrations are used to measure the excretory capacity of the kidneys, accumulation of these two molecules themselves do not account for the many symptoms and signs that characterize the uremic syndrome in advanced renal failure. Hundreds of toxins that accumulate in renal failure have been implicated in the uremic syndrome. These include water-soluble, hydrophobic, protein-bound, charged, and uncharged compounds. Additional categories of nitrogenous excretory products include guanidino compounds, urates and hippurates, products of nucleic acid metabolism, polyamines, myoinositol, phenols, benzoates, and indoles. Compounds with a molecular mass between 500 and 1500 Da, the so-called middle molecules, are also retained and contribute to morbidity and mortality. It is thus evident that the serum concentrations of urea and creatinine should be viewed as being readily measured, but incomplete, surrogate markers for these compounds, and monitoring the levels of urea and creatinine in the patient with impaired kidney function represents a vast oversimplification of the uremic state.

Clinical Features Chronic Kidney Disease And Uremia

Uremia leads to disturbances in the function of virtually every organ system. Chronic dialysis can reduce the incidence and severity of many of these disturbances, so that the overt and florid manifestations of uremia have largely disappeared in the modern health setting.

Fluid and electrolyte disturbances: Volume expansion, Hyponatremia, Hyperkalemia and Hyperphosphatemia.

Endocrine-metabolic disturbances: Secondary hyperparathyroidism, Adynamic bone, Vitamin D–deficient, osteomalacia, Carbohydrate resistance, Hyperuricemia, Hypertriglyceridemia, Increased Lp(a) level, Decreased high-density lipoprotein level, Protein-energy malnutrition, Impaired growth and development, Infertility and sexual dysfunction, and Amenorrhea.

Neuromuscular disturbances: Fatigue, Sleep disorders, Headache, Impaired mentation, Lethargy , Asterixis, Muscular irritability, Peripheral neuropathy, Restless legs syndrome, Myoclonus, Seizures, Coma, Muscle cramps, Dialysis disequilibrium syndrome and Myopathy.

Cardiovascular and pulmonary disturbances: Arterial hypertension, Congestive heart failure or pulmonary edema, Pericarditis, Hypertrophic or dilated cardiomyopathy, Uremic lung, Accelerated atherosclerosis, Hypotension and arrhythmias and vascular calcification.

Dermatologic disturbances: Pallor, Hyperpigmentation, Pruritus, Ecchymoses, Nephrogenic fibrosing, dermopathy and Uremic frost.

Gastrointestinal disturbances: Anorexia, Nausea and vomiting, Gastroenteritis, Peptic ulcer, Gastrointestinal bleeding, Idiopathicascites and Peritonitis.

Hematologic and immunologic disturbances: Anemia, Lymphocytopenia, Bleeding diathesis, increased susceptibility to infection, Leukopenia and Thrombocytopenia.

History and Physical Examination

Symptoms and overt signs of kidney disease are often subtle or absent until renal failure supervenes. Thus, the diagnosis of kidney disease often surprises patients and may be a cause of skepticism and denial. Particular aspects of the history that are germane to renal disease include a history of hypertension (which can cause CKD or more commonly be a consequence of CKD), diabetes mellitus, abnormal urinalyses, and problems with pregnancy such as preeclampsia or early pregnancy loss. A careful drug history should be elicited: patients may not volunteer use of analgesics, for example. Other drugs to consider include nonsteroidal anti-inflammatory agents, gold, penicillamine, antimicrobials, chemotherapeutic agents, antiretroviral agents, proton pump inhibitors, phosphate-containing bowel cathartics, and lithium, as well as prior exposure to medical imaging radiocontrast agents. In evaluating the uremic syndrome, questions about appetite, weight loss, nausea, hiccups, peripheral edema, muscle cramps, pruritus, and restless legs are especially helpful. A careful family history of kidney disease, together with assessment of manifestations in other organ systems such as auditory, visual, integumentary and others, may lead to the diagnosis of a heritable form of CKD (e.g., Alport or Fabry syndrome, cystinuria, among others) or shared environmental exposure to nephrotoxic agents (e.g., heavy metals, aristolochic acid). It should be noted that clustering of CKD, sometimes of different etiologies, is often observed within families.

Physical examination

The physical examination should focus on blood pressure and target organ damage from hypertension. Thus, funduscopy and precordial examination (left ventricular heave, a fourth heart sound) should be carried out. Funduscopy is important in the diabetic patient, as it may show evidence of diabetic retinopathy, which is associated with nephropathy. Other physical examination manifestations of CKD include edema and sensory polyneuropathy. The finding of asterixis or a pericardial friction rub not attributable to other causes usually signifies the presence of the uremic syndrome.

Ancillary Investigations

Laboratory studies should focus on a search for clues to an underlying causative or aggravating disease process and on the degree of renal damage and its consequences. Serum and urine protein electrophoresis, looking for multiple myeloma, should be obtained in all patients >35 years with unexplained CKD, especially if there is associated anemia and elevated, or even inappropriately normal, serum calcium concentration in the face of renal insufficiency. In the presence of glomerulonephritis, autoimmune diseases such as lupus and underlying infectious etiologies such as hepatitis B and C and HIV should be assessed. Serial measurements of renal function should be obtained to determine the pace of renal deterioration and ensure that the disease is truly chronic rather than acute or subacute and hence potentially reversible. Serum concentrations of calcium, phosphorus, vitamin D, and PTH should be measured to evaluate metabolic bone disease. Hemoglobin concentration, iron, B12, and folate should also be evaluated. A 24-h urine collection may be helpful, as protein excretion >300 mg may be an indication for therapy with ACE inhibitors or ARBs.

Imaging Studies

The most useful imaging study is a renal ultrasound, which can verify the presence of two kidneys, determine if they are symmetric, provide an estimate of kidney size, and rule out renal masses and evidence of obstruction. The diagnosis of renovascular disease can be undertaken with different techniques, including Doppler sonography, nuclear medicine studies, or CT or MRI studies. If there is a suspicion of reflux nephropathy (recurrent childhood urinary tract infection, asymmetric renal size with scars on the renal poles), a voiding cystogram may be indicated. However, in most cases by the time the patient has CKD, the reflux has resolved, and even if still present, repair does not improve renal function. Radiographic contrast imaging studies are not particularly helpful in the investigation of CKD. Intravenous or intra arterial dye should be avoided where possible in the CKD patient, especially with diabetic nephropathy, because of the risk of radiographic contrast dye–induced renal failure. When unavoidable, appropriate precautionary measures include avoidance of hypovolemia at the time of contrast exposure, minimization of the dye load, and choice of radiographic contrast preparations with the least nephrotoxic potential. Additional measures thought to attenuate contrast-induced worsening of renal function include judicious administration of sodium bicarbonate–containing solutions and N-acetyl-cysteine.

Renal Biopsy

In the patient with bilaterally small kidneys, renal biopsy is not advised because (1) it is technically difficult and has a greater likelihood of causing bleeding and other adverse consequences, (2) there is usually so much scarring that the underlying disease may not be apparent, and (3) the window of opportunity to render disease-specific therapy has passed. Other contraindications to renal biopsy include uncontrolled hypertension, active urinary tract infection, bleeding diathesis (including ongoing anticoagulation), and severe obesity. Ultrasound-guided percutaneous biopsy is the favored approach, but a surgical or laparoscopic approach can be considered, especially in the patient with a single kidney where direct visualization and control of bleeding are crucial. In the CKD patient in whom a kidney biopsy is indicated (e.g., suspicion of a concomitant or superimposed active process such as interstitial nephritis or in the face of accelerated loss of GFR), the bleeding time should be measured, and, if increased, desmopressin should be administered immediately prior to the procedure.

MCQs

1. Which of the following statements is accurate regarding the presentation of nephrotic syndrome
   a. Hematuria is among the most common presenting symptoms in adults with nephrotic syndrome
   b. The first sign of nephrotic syndrome in children is usually swelling of the face
   c. The presence of deep venous thrombosis or pulmonary embolism suggests a diagnosis other than nephrotic syndrome
   d. Weight loss and hypotension are frequently present in adults with nephrotic syndrome
2. Which of the following is accurate regarding BPH and prostate-related issues?
   a. All men with BPH demonstrate lower urinary tract symptoms (LUTS), and all men with LUTS have BPH
   b. The risk for acute urinary retention (AUR) increases with age
   c. Prostate volume typically decreases over time in men with BPH
   d. Complications (eg, bladder stones or recurrent urinary tract infections [UTIs]) are frequent
3. Of the following, which is more commonly recognized as a secondary cause of nephrotic syndrome
   a. SjĨgren syndrome b. Cushing disease
   c. Hemolytic anemia d. Amyloidosis
4. In distinguishing acute from chronic renal failure, which of the following is the most reliable evidence of CKD?
   a. History of increased serum creatinine

b. History of increased BUN

c. Above-normal plasma sodium

d. Above-normal serum calcium

5. Which statement regarding the diagnosis of CKD is most accurate?

a. Normochromic normocytic anemia typically begins in late-stage CKD

b. Measurement of GFR alone is sufficient for identifying stage 1 and stage 2 CKD

c. Hypertension is typically a marker of CKD

d. eGFR lacks precision and accuracy until GFR reaches < 60 mL/min/1.73 m2

6. The severity of BPH assessment is done by

a. International Prostate Symptom Score (IPSS)

b. American nephrological Association Symptom Index (ANA-SI)

c. Life quality scale

d. Quality of life (QOL)

Answer

1.	b	2.	b	3.	d
4.	a	5.	d	6.	a

CHAPTER 12

Hikka Roga

Introduction

This is a state of intermittent spasm of diaphragm accompanied by sudden closure of glottis. It may be caused by stimulation of sensory nerve endings. Vitiation of *Udāna* and *Prāṇa Vāyu* produces outlet of *Vāyu* with 'hik-hik' sound from the mouth with a spasm of the abdomen. This indisposition is identified as *Hikkā*. It is of five types viz. *Ānnaja, Yamalā, Kṣudrā, Gambhīrā* and *Mahatī. (AH. Ni.* 4. 18*; CS. Ci.* 17. 10-13*; SS. Ut.* 50. 3-4*) Ban. hikkā, Guj. hedkī, Hin. hicakī, Kan. ikkdu, Mal. hikkle, ikkiṭṭam, Mar. uchakī, Ori. hakuṭī, Pan. hickī, Tam. vikkal, Tel. ikkillū.*

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

हिक्कातेऽनयेति हिक्काः ।*Śabdakalpdruma*

That which produces a spasmodic sound in the throat is called *Hikkā.*

हिक्कति कूजत्यनया हिक्काः । *Śabdakalpdruma*

That which produces an indistinct sound is called *Hikkā.*

हिक्कयते हिनस्तोति वा ।*Śabdakalpdruma*

It debilitate the individual due to its effect is called *Hikkā.*

हिगिति कृन्ना कायति शब्दायते इति ॥

M.Ni. 12/3 (Madhu)

तत्र हिगिति कायति शब्दं करोति हिक्का ॥

C.Ci. 17/27 (Cakrapaṇi)

Hiccups are repeated spasms of the diaphragm paired with a 'hic' sound from the vocal cords closing are called *Hikkā.*

हिक्कास्वरूपं तु घोषवत्त्वेन प्रतिपादितं ॥

Āḍd.Śa.Prā. 7/25

The term *Hikkā*/hiccup derives from the sound of the event.

मुहुर्मुहर्वायुरुदेति सस्वनो यकृत्प्लीहान्त्राणि मुखादिवाक्षिपन् ।
स घोषवानाशु हिनस्ति यस्मात् ततस्तु हिक्केत्यभिघीयते बुधैः इति ॥

Āḍd.Śa.Prā. 7/25

मुहुर्मुहर्वायुरुदेति सस्वनो यकृत्प्लीहान्त्राणि मुखादिवाक्षिपन् ।
स घोषवानाशु हिनस्त्यसून् यतस्ततस्तु हिक्केति भिषग्भिरुच्यते ॥

S.S.Utt. 50/6

When *Vāyu* repetitively and intensely brings out a specific sound *'Hik'* through the mouth by pulling *Yakṛt* (liver), *Plīha* (spleen) and *Antrāṇi* (intestines), and is fatal in nature is called *Hikkā* by the physicians.

हिक्का रोग निदान (Nidāna of Hikkā Roga)

पृथक् पञ्चविधावेतौ निर्दिष्टौ रोगसंग्रहे ।
तयोः शृणु समुत्थानं लिङ्गं च सभिषग्जितम् ॥
रजसा धूमवाताभ्यां शीतस्थानाम्बुसेवनात् ।
व्यायामाद्ग्राम्यधर्माध्वरुक्षान्नविषमाशनात् ॥
आमप्रदोषादानाहाद्रोक्ष्यादत्यपतर्पणात् ।
दौर्बल्यान्मर्मणो घातादृद्वन्द्वाच्छुद्धयतियोगतः ॥
अतीसारज्वरच्छर्दिप्रतिश्यायक्षतक्षयात् ।
रक्तपित्तादुदावर्ताद्वि सूच्यलसकादपि ॥
पाण्डुरोगाद्विषाच्चैव प्रवर्तेते गदाविमौ ।
निष्पावमाषपिण्याकतिलतैलनिषेवणात् ॥
पिष्टशालूकविष्टम्भिविदाहिगुरुभोजनात् ।
जलजानूपपिशितदध्यामक्षीरसेवनात् ॥
अभिष्यन्द्युपचाराच्च श्लेष्मलानां च सेवनात् ।
कण्ठोरसः प्रतीघाताद्विबन्धैश्च पृथग्विधैः ॥

C.S.Ci. 17/10-16

विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः ।
शीतपानासनस्थानरजोधूमानिलानलैः ॥
व्यायामकर्मभाराध्ववेगाघातापतर्पणैः ।
आमदोषाभिघातस्त्रीक्षयदोषप्रपीडनैः ॥
विषमाशनाध्यशनैस्तथा समशनैरपि ।
हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥

S.S.Utt. 50/3-5

The following etiological factors manifest Śvāsa and Hikkā Roga :- Exposure to dust, smoke, fire and wind; residing in a cold place & use of cold water; physical exertion;, sexual intercourse & long walk beyond one's capacity; intake of rough food; irregular meals; manifestation of certain reactions inside the body due to underprocessed *Anna Rasa* (*Āma Pradoṣa*); distension of abdomen; roughness; pasting in excess; debility; injury to vital organs; usage of mutually contradictory substances; excessive administration of biopurifacatory therapies.

Regular consumption of *Niṣpāva*, black gram, oilcake, sesame oilconsumption of flour preparations, tubers, constipating & burning substances, heavy food; intake of meat of aquatic & marshy animals & birds; intake of curd & unboiled milk; consumption of channel blocking substances; regular intake of *Kapha* aggravating factors; injury to throat & chest and various other sorts of retention and suppression of natural urges.

These diseases manifest as complications in following diseases: *Atisāra, Jvara, Chardi, Pratiśyāya, Kṣata, Kṣaya, Raktapitta, Udāvarta, Visūcikā, Alasaka, Pāṇḍu & Viṣa.*

हिक्का रोग सम्प्राप्ति (Samprāpti of Hikkā Roga)

विहाय प्रकृति वायुः प्राणोऽथ कफसंयुतः।
श्वासयत्यूदूर्ध्वगो भूत्वा तं श्वासं परिचक्षते॥
S.S.Utt. 51/4

मारुतः प्राणवाहीनि स्त्रोतांस्याविश्य कुप्यति।
उरःस्थः कफमुद्धूय हिक्काश्वासान् करोति सः॥
घोरान् प्राणोपरोधाय प्राणिनां पञ्च पञ्च च।
C.S.Ci. 17/17-18

यदा स्त्रोतांसि संरुध्य मारुतः कफपूर्वकः।
विष्वग्व्रजति संरुद्धस्तदा श्वासान्करोति सः॥
C.S.Ci. 17/45

Due to consumption of etological factors *Prāṇavāta* combining with *Kapha* leaves its own path and begins to move in the upward direction, giving rise to more of respirations is known as *Śvāsa Roga* & *Hikkā Roga*, which are of five kinds each & which may lead to the death of the patient.

कफवातात्मकावेतौ पित्तस्थानसमुद्भवौ।
हृदयस्य रसादीनां धातूनां चोपशोषणौ॥
तस्मात् साधारणावेतौ मतौ परमदुर्जयौ।
मिथ्योपचरितौ क्रुद्धौ हत आशीविषाविव॥
C.S.Ci. 17/8-9

Both *Hikkā* & *Śvāsa* originates from the seat of *Pitta* and are caused by the simultaneous aggravation of *Kapha* & *Vāta* and dries up the *Hṛdaya* & *Rasādi Dhātus*. Hence both being similar are regarded as very difficult to treat and if treated inappropriately leading to further aggravation of the condition & kill the patient like serpents.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphadi doṣās*
↓
Development of *Āma* inside the gastro intestinal tract and *Śvāsa* originates from the seat of *Pitta*
↓
Localisation of *Kapha doṣa* (especially *Rasadhātugata Malarupi Kapha)* in the *Prāṇavaha Srotas*
↓
Leading to aggravation of *Vāta doṣa* inside the *Prāṇavaha Srotas* leaves its own path and begins to move in the upward direction
↓
Dries up the *Hṛdaya* & *Rasādi Dhātus.*
↓
Hikkā Roga

प्राणोदकान्नवाहीनि स्त्रोतांसि सकफोऽनिलः।
हिक्काः करोति संरुध्य तासां लिङ्गं पृथक् शृणु॥
C.S.Ci. 17/21

Both *Vāta* and *Kapha* gets obstructed in *Prāṇavaha, Udakavaha & Annavaha Srotas* and manifest *Hikkā Roga.*

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphadi doṣās*
↓

Development of *Āma* inside the gastro intestinal tract and *Śvāsa* originates from the seat of *Pitta*

↓

Localisation of *Kapha doṣa* (especially *Rasadhātugata Malarupi Kapha)* in the *Prāṇavaha Srotas*

↓

Leading to aggravation of *Vāta doṣa* in side the *Prāṇavaha Srotas* leaves its own path and begins to move in the upward direction

↓

Both *Vāta* and *Kapha* gets obstructed in *Prāṇavaha, Udakavaha & Annavaha Srotas*

↓

Hikkā Roga

हिक्का रोग सम्प्राप्ति घटक (Samprāpti Ghaṭaka of Hikkā Roga)

Doṣa : *Vāta-Kapha* (*Prāṇa* and *Udāna Vāta, Bhodaka & Kledaka Kapha*)
Dūṣya : *Dhātu → Rasa Dhātu*
Upadhātu →
Śārīrika mala → Mūtra, Puriṣa
Dhātumala→Kapha, Pitta, Tvacā and *Sveda*
Agni : *Jaṭharāgni, Dhātvāgni*
Agnidușṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Prāṇavaha, Udakavaha, Annavaha and Rasavaha*
Srotoduṣṭi :*Saṅga & Vimārgamana*
Udbhavasthāna : *Āmāśayottha (Pittasthāna)*
Vyaktasthāna : *Āsya, Grīvā, Pṛṣṭa, Pārśva*
Sancārasthāna : *Prāṇavaha Sroto Avayava*
Adhiṣṭhāna : *Pitta Sthāna*
Prabhāva : *Kṣudra Śvāsa→Sādhya*
Tamaka Śvāsa→ Kṛcchra Sādhya Mahā, Cinna and Urdvaśvāsa→ Asādhya
Svabhāva : *Āśukāri*
Rogamārga : *Madhyama Roga Mārga*
Roga Bheda : 5 kinds

पूर्वरूप (Pūrvarupa)

कण्ठोरसोर्गुरुत्वं च वदनस्य कपायता।
हिक्कानां पूर्वरुपाणि कुक्षेराटोप एव च॥
C.S.Ci. 17/19

मुखं कपायमरतिर्गौरवं कण्ठवक्षसोः।
पूर्वरूपाणि हिक्कानामाटोपो जठरस्य च॥
S.S.Utt. 50/8

Premonitory signs and symptoms of Hikkā are as follows:- Feeling of heaviness in the throat & chest, astringent taste in the mouth, *Āṭopa* and restlessness.

महाहिक्का (Mahāhikkā)

क्षीणमांसबलप्राणतेजसः सकफोऽनिलः।
गृहीत्वा सहसा कण्ठमुच्चैर्घोषवतीं भृशम्॥
करोति सततं हिक्कामेकद्वित्रिगुणां तथा।
प्राणः स्त्रोतांसि मर्माणि संरुध्योष्माणमेव च॥
संज्ञा मुष्णाति गात्राणां स्तम्भं संजनयत्यपि।
मार्गं चैवान्नपानानां रुणद्ध्युपहतस्मृतेः॥
साश्रुविप्लुतनेत्रस्य स्तब्धशङ्खच्युतभ्रुवः।
सक्तजल्पप्रलापस्य निर्वृतिं नाधिगच्छतः॥
महामूला महावेगा महाशब्दा महाबला।
महाहिक्केति सा नृणां सद्यः प्राणहरा मता॥
इति महाहिक्का। *C.S.Ci. 17/22-26*

मर्माण्यापीडयन्तीव सततं या प्रवर्तत॥
देहमायम्य वेगेन घोषयत्यतितृष्यतः।
महाहिक्केति सा ज्ञेया सर्वगात्रप्रकम्पिनी॥
S.S.Utt. 50/13-14

Vāta in association with *Kapha* suddenly afflicts the throat in those patients who is suffering from depletion of *Māmsa, Bala, Prāṇa & Tejas* causes constant hiccup with excessively high sound, which is characterised by one, two or three bouts at a time with continuity. *Prāṇavāta* seizes the functions of *Srotas, Marmās* and *Agni* of the patient leading to unconsciousness, stiffness of body parts and restricts the proper entry of foods & drinks. Patient also suffers from loss of memory, profuse lacrymation in the eyes, stiffness in temples, drooping of eyebrows, mingled voice, delirium and doesn't find comfort by any means. *Mahāhikkā* having deep root, forceful paroxysms, loud sound and severe and which kills the patient instantaneously. *Suśruta* mentioned profound thirst and tremors all over the body. It is termed as *Mahāhikkā* because patient produces exceedingly loud sound.

गम्भीर हिक्का (Gambhīra Hikkā)

हिक्कते यः प्रवृद्धस्तु कृशो दीनमना नरः।
जर्जरीणोरसा कृच्छ्रं गम्भीरमनुनादयन्॥
संजृम्भन् संक्षिपंश्चैव तथाऽङ्गानि प्रसारयन्।
पार्श्वे चोभे समायम्य कूजन् स्तम्भरुगर्दितः॥
नाभेः पक्वाशयाद्वापि हिक्का चास्योपजायते।
क्षोभयन्ती भृशं देहं नामयन्तीव ताम्यतः॥
रुणद्ध्युच्छ्वासमार्गं तु प्रणष्टबलचेतसः।
गम्भीरा नाम सा तस्य हिक्का प्राणान्तिकी मता॥
इति गम्भीरा हिक्का। *C.S.Ci. 17/27-30*

नाभिप्रवृत्ता या हिक्का घोरा गम्भीरनादिनी।
शुष्कौष्ठकण्ठजिह्वास्यश्वासपार्श्वरुजाकरी॥
अनेकोपद्रवयुता गम्भीरा नाम सा स्मृता॥
S.S.Utt. 50/12-13

It is the advanced stage of *Hikkā* in which patient hiccups frequently & excessively and produces deep, painful and resonant sound with his shattered chest. It is associated with yawning, contraction & expansion of body parts espescially limbs, produces indistinct sound while holding his both flanks associated with stiffness & pain, it starts from navel & gastric regions causing excessive jerks, bending of body, feeling of darkness and obstructs the respiratory passage with loss of strength & consciousness. It is known as *Gambhīra Hikkā,* which is fatal. Patient also suffers from dryness of the lips, throat, tongue & mouth, dyspnoea, pain in the flanks & associated with many secondary diseases.

व्यपेता हिक्का (Vyapetā Hikkā)

व्यपेता जायते हिक्का याऽन्नपाने चतुर्विधे।
आहारपरिणामान्ते भूयश्च लभते बलम्॥
प्रलापवम्यतीसारतृष्णार्तस्य विचेतसः।
जृम्भिणो विप्लुताक्षस्य शुष्कास्यस्य विनामिनः॥
पर्याध्मातस्य हिक्का या जत्रुमूलादसन्तता।
सा व्यपेतेति विज्ञेया हिक्का प्राणोपरोधिनी॥
इति व्यपेता हिक्का। *C.S.Ci. 17/31-33*

Disease *Vyapetā Hikkā* ensues during the course of the intake of four types of foods & drinks and gets aggravated after the digestion of food. It is characterised by delirium, vomiting, diarrhoea, morbid thirst, mental confusion, yawning, congestion of eyes, dryness of mouth, bending of the body, severe tympanitis. The *Hikkā* originating off & on from the root of the neck is known as *Vyapetā Hikkā* and it is obstructing the vital breath.

क्षुद्र हिक्का (Kṣudra Hikkā)

क्षुद्रवातो यदा कोष्ठाद्व्यायामपरिघट्टितः।
कण्ठे प्रपद्यते हिक्कां तदा क्षुद्रां करोति सः॥
अतिदुःखा न सा चोरःशिरोमर्मप्रबाधिनी।
न चोच्छ्वासान्नपानानां मार्गमावृत्य तिष्ठति॥
वृद्धिमायस्यतो याति भुक्तमात्रे च मार्दवम्।
यतः प्रवर्तते पूर्वं तत एव निवर्तते॥
हृदयं क्लोम कण्ठं च तालुकं च समाश्रिता।
मृद्वी सा क्षुद्रहिक्केति नृणां साध्या प्रकीर्तिता॥
C.S.Ci. 17/34-37

विकृष्टकालैर्या वेगैर्मन्दैः समभिवर्तते।
क्षुद्रिका नाम सा हिक्का जत्रुमूलात् प्रधाविता॥
S.S.Utt. 50/11-12

During the course of physical exertion *Vāta* is slightly aggravated moves from belly to throat causes the *Kṣudra Hikkā.* It doesn't cause trouble to the patient, neither it afflicts the vital parts of chest nor it obstruct the passage of respiration & food. It gets aggravated after physical exercises and subsides after meal in the same way as it starts. It is located in the heart, *Kloma,* throat & palate. It is of mild in nature & which is curable is known as *Kṣudra Hikkā.* It appears at any time, with mild bouts & arises from the root of the neck.

अन्नज हिक्का (Annaja Hikkā)

सहसाऽत्यभ्यवहृतैः पानान्नैः पीडितोऽनिलः।
ऊर्ध्वं प्रपद्यते कोष्ठान्मद्यैर्वाऽतिमदप्रदैः॥
तथाऽतिरोषभाष्याध्वहास्यभारातिवर्तनैः।
वायुः कोष्ठगतो धावन् पानभोज्यप्रपीडितः॥
उरःस्रोतः समाविश्य कुर्याद्धिक्कां ततोऽन्नजाम्।
तथा शनैरसंबन्धं क्षुवंश्चापि स हिक्कते॥
न मर्मबाधाजननी नेन्द्रियाणां प्रबाधिनी।

हिक्का पीते तथा भुक्ते शमं याति च साऽन्नजा ॥
इत्यन्नजा हिक्का । *C.S.Ci. 17/38-41*

त्वरमाणस्य चाहारं भुञ्जानस्याथवा घनम् ।
वायुरन्नैरवस्तीर्णः कटुकैरदितो भृशम् ।
हिक्वयत्यूर्ध्वगो भूत्वा तां विद्यादन्नजां भिषक् ॥
S.S.Utt. 50/9

Vāta gets aggravated due to sudden intake of drinks & food, in large quantity, eating solid & pungent foods, consumption of intoxicating alocohol products, excessive anger, speech, long walk, laughing for longer period of time and carrying heavy weight and moves upwards from belly and enters the channels of the chest & produces *Annaja Hikkā*. It is characterised by slow hiccups and with interruptions even while he sneezes. It does not afflict the parts and sensory organs. It gets subsided by the intake of foods & drinks. It is known as *Annaja Hikkā.*

यमिका हिक्का (Yamikā Hikkā) Or यमला हिक्का (Yamalā Hikkā)

अतिसंचितदोषस्य भक्तच्छेदकृशस्य च ।
व्याधिभिः क्षीणदेहस्य वृद्धस्यातिव्यवायिनः ॥
आसां या सा समुत्पन्ना हिक्का हन्त्याशु जीवितम् ।
यमिका च प्रलापार्तितृष्णामोहसमन्विता ॥
अक्षीणश्चाप्यदीनश्च स्थिरधात्विन्द्रियश्च यः ।
तस्य साधयितुं शक्या यमिका हन्त्यतोऽन्यथा ॥
C.S.Ci. 17/42-44

चिरेण यमलैर्वेगैर्या हिक्का सम्प्रवर्त्तते ।
कम्पयन्ती शिरोग्रीवं यमलां तां विनिर्दिशेत् ॥
S.S.Utt. 50/10-11

Yamikā Hikkā arises in those individuals who is having great accumulation of *Doṣās,* weakness due to discontinuation of food, emaciated due to diseases, old age, excessive indulgence of sex and which can kill the patient quickly. It is characterised by *Hikkā* which comes in pairs after long periods of time, causing shaking of head & neck, delirium, distress, thirst, unconsciousness. It is curable if the patient is not emaciated, anxious & possesses firm *Dhātus* & sense organs, otherwise it is fatal.

साध्यासाध्या (Sādhyāsādhyatā)

Mahāhikkā, Gambhīra Hikkā & Vyapetā Hikkā are incurable.

Kṣudra Hikkā & Annaja Hikkā are curable.

आयम्यते हिक्कतोऽङ्गानि यस्य दृष्टिश्चोर्ध्वं ताम्यते यस्य गाढम् ।
क्षीणोऽन्नद्विद् कासते यश्च हिक्की तौ द्वावन्त्यौ वर्जयेद्धिक्कमानौ ॥
S.S.Utt. 50/15

One who expands his body during bouts of *Hikkā,* who has his gaze fixed upwards, becomes unconscious deeply, one who is emaciated, dislikes food & coughing frequently should be rejected for treatment. *Gambhīra & Mahāhikkā* should be refused for treatment.

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Harmful activities and items in cases of Hikkā roga includes : Suppression of the natural urges, nasal therapies, the *Vasti* therapy, chewing wood sticks with the teeth, hard labour, excessive walking, lifting heavy items, facing dust, moving out in sun, consuming meals heavy to digest, having intercourse, taking hot items of meal, taking meat of the animals from the marshy region, taking deep fried nuggets and the like dishes, taking items that produce *Kapha*, taking the *Urada* beans, blood-letting, facing the easternly wind, drinking water immediately after eating meals, taking the milk and *Ghṛta* of sheep, drinking impure water, eating fishes, vegetable leaves of *Kanda* and the like tubers, leaves of mustard, etc. (B.R. 16/137-139)

Pathya and Apathya-Hikkā Roga

उपशय/पथ्य (Upaśaya/Pathya)

Useful items and activities in cases of *Hikkā* . Sweating, vomiting, smoking herbal preparations, sleeping, oily eatables, salt in limited quantity, pulse of *Kulattha*, old wheat, old red variety of *Śāli* rice, old barley, garlic bulbs, *Paṭola* (small cucumber-snake gourd), radish, basil seeds, honey, cow-urine, breathing exercises, smelling the steam coming by the dropping water on burning soil, are very useful in cases of hiccough. (B.R. 16/122-128)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Avoidable items and activities in cases of *Hikkā.*

Suppression natural urges, facing dust, wind, hot sun, hard excercises, incomptiable meals, constipative, hot, sour and cough-generating eatables, *Niṣpāva*, paste-made eatables, *Urada* beans, cakes of sesame, flesh of birds from marshy regions, milk of sheep, fish, leaves of mustard, citrus items, potato chips deep fried in oil, leaves of *Kanda* and like tubers, mint and cold drinks etc. are harmful for the patients suffering from hiccough. Similarly, the *Basti* therapy and use of tooth-twigs are to be avoided by the patient. (B.R. 16/129-131)

MCQs

1. आहारपरिणामान्तेभूयश्चलभतेबलम is clinical feature of
   a. व्यपेताहिक्का (Vyapeta Hikka)
   b. अन्नजाहिक्का (Annaja Hikka)
   c. गम्भीराहिक्का (Gambhira Hikka)
   d. क्षुद्राहिक्का (Kshudra Hikka)
2. *Hikka* having *Jatrumoola* as *udbhavasthana*
   a. *Maha Hikka* b. *Kshudra Hikka*
   c. *Gambhira Hikka* d. *Annaja Hikka*
3. *Marmanyutpidayanti* is the feature observed in
   a. *Gambhira Hikka* b. *Annaja Hikka*
   c. *Maha Hikka* d. *Kshudra Hikka*
4. *Vadanasyakashayata* is the *purvaroopa* of
   a. *Swasa* b. *Kasa*
   c. *Ajeerna* d. *Hikka*
5. Types of *Hikka* according to *Ashtanga Hridaya* include all except
   a. *Bhaktodbhava* b. *Vyapetha*
   c. *Yamala* d. *Gambhir Hikka*
6. आमादोषाभिघातस्त्रीक्षयदोषप्रपीडनैः । विषमाशनाध्यनशनैस्तथा समशनैरपि are the etiological factors responsible for
   a. *Hikka swasha kasa* b. *Swarabheda*
   c. *Urahkshata* d. *Rajayakshma*
7. *Ghoran Pranoparodhaya praninam pancha pancha mentioned in context to*
   a. *Urahkshata* b. *Hikka & Swasha*
   c. *Rajayakshma* d. *Kasa*
8. *Sarvagatraprakampini* is the feature of
   a. *Gambhira Hikka* b. *Annaja Hikka*
   c. *Maha Hikka* d. *Kshudra Hikka*
9. *Nabhipravritta ya Hikka mentioned in context to*
   a. *Maha Hikka* b. *Kshudra Hikka*
   c. *Gambhira Hikka* d. *Annaja Hikka*
10. Kampayanti shirogrivam *mentioned in context to*
    a. *Gambhira Hikka* b. *Yamika Hikka*
    c. *Maha Hikka* d. *Kshudra Hikka*
11. Aharaparinamante bhuyascha labhate balam *mentioned in context to*
    a. *Annaja Hikka* b. *Yamika Hikka*
    c. *Maha Hikka* d. *Vyapeta Hikka*

Answer

1.	a	2.	b	3.	c
4.	d	5.	b	6.	a
7.	b	8.	c	9.	c
10.	b	11.	d		

CHAPTER
13

Shwasa Roga

श्वास रोग (Śvāsa Roga)

Introduction

There are innumerable number of diseases classifiable into two categories i : e *Sāmānya* & *Asāmānya* produced by the aggravation of three *Doṣās* as a result of three etiological factors. These are deadly diseases like *Sannipātika Jvara, Śaṅkhaka, Rohiṇi* etc. which can kill the patient. But none of these is as deadly as *Śvāsa* & *Hikkā* that can kill a patient instanteneously. Even if he has been suffering from various ailments, ultimately he falls a victim of *Śvāsa* & *Hikkā* that are immensly painful.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

Svāsa term indicates difficulty in breathing or shortness of breath. It is a symptom of many conditions that affect the *Prāṇvaha* srotas (respiratory system).

श्वसित्यनेनेति श्वासः ॥ *Śabdakalpdruma*
श्वसितौति श्वासः ॥ *Śabdakalpdruma*
कासः कुत्सित शब्दः ॥ *Vācaspatyam*

That which produces 'sigh' is called *Śvāsa*

श्वास इति वेगवदूर्ध्ववातन्नं ॥ *Āḍd.Śa.Prā. 7/245*

The disease which originates from the forceful upward movement of *Vāta* is called *Śvāsa*

विहाय प्रकृतिं वायुः प्राणोऽथ कफसंयुतः ।
श्वासयत्यूर्ध्वगो भून्ना तं श्वासं परिचक्षते ॥
Su.Utt. 51/4

प्राणो वायुः प्रकृतिं विहाय विगुणो भून्नेत्यर्त्थः, ऊर्ध्वगो भून्ना तथा कफसंयुतः सन् यदा श्वासयति तं बुधाः श्वासं परिचक्षते ॥

Su.Utt. 51/4 (Dalhaṇa)

The aggravated *Prāṇa Vāta* combines with *Kapha* moves *Ūrdhva Gati* (upwards) and produces gasping and laboured breathing which is called *Svāsa*.

श्वास रोग निदान (Nidāna of Śvāsa Roga)

पृथक् पञ्चविधावेतौ निर्दिष्टौ रोगसंग्रहे ।
तयोः शृणु समुत्थानं लिङ्गं च सभिषग्जितम् ॥
रजसा धूमवाताभ्यां शीतस्थानाम्बुसेवनात् ।
व्यायामाद्ग्राम्यधर्माध्वरुक्षान्नविषमाशनात् ॥
आमप्रदोषादानाहाद्रोक्ष्यादत्यपतर्पणात् ।
दौर्बल्यान्मर्मणो घातादद्वन्द्वाच्छुद्ध्यतियोगतः ॥
अतीसारज्वरच्छर्दिप्रतिश्यायक्षतक्षयात् ।
रक्तपित्तादुदावर्ताद्विसूच्यलसकादपि ॥
पाण्डुरोगाद्विषाच्चैव प्रवर्तेते गदाविमौ ।
निष्पावमाषपिण्याकतिलतैलनिषेवणात् ॥
पिष्टशालूकविष्टम्भिविदाहिगुरुभोजनात् ।
जलजानूपपिशितदध्यामक्षीररसेवनात् ॥
अभिष्यन्द्युपचाराच्च श्लेष्मलानां च सेवनात् ।
कण्ठोरसः प्रतीघाताद्विबन्धैश्च पृथग्विधैः ॥
C.S.Ci. 17/10-16

विदाहिगुरुविष्टम्भिरूक्षाभिष्यन्दिभोजनैः ।
शीतपानासनस्थानरजोधूमानिलानलैः ॥
व्यायामकर्मभाराध्ववेगाघातापतर्पणैः ।
आमदोषाभिघातस्त्रीक्षयदोषप्रपीडनैः ॥
विषमाशनाध्यशनैस्तथा समशनैरपि ।
हिक्का श्वासश्च कासश्च नृणां समुपजायते ॥
S.S.Utt. 50/3-5

The following etiological factors manifest Śvāsa and Hikkā Roga :- Exposure to dust, smoke, fire and wind; residing in a cold place & use of cold water; physical exertion;, sexual intercourse & long walk beyond one's capacity; intake of rough food; irregular meals; manifestation of certain reactions inside the body due to underprocessed *Anna Rasa* (*Āma Pradoṣa*); distension of abdomen; roughness; fasting in excess; debility; injury to vital organs; usage of mutually contradictory substances; excessive administration of biopurifacatory therapies.

Regular consumption of *Niṣpāva*, black gram, oilcake, sesame oilconsumption of flour preparations, tubers, constipating & burning substances, heavy food; intake of meat of aquatic

& marshy animals & birds; intake of curd & unboiled milk; consumption of channel blocking substances; regular intake of *Kapha* aggravating factors; injury to throat & chest and various other sorts of retention and suppression of natural urges.

These diseases manifest as complications in following diseases: *Atisāra, Jvara, Chardi, Pratiśyāya, Kṣata, Kṣaya, Raktapitta, Udāvarta, Visūcikā, Alasaka, Pāṇḍu & Viṣa.*

श्वास रोग संप्राप्ति (Samprāpti of Śvāsa Roga)

विहाय प्रकृति वायुः प्राणोऽथ कफसंयुतः।
श्वासयत्यूद्ध्वंगो भूत्वा तं श्वासं परिचक्षते॥
S.S.Utt. 51/4

मारुतः प्राणवाहीनि स्त्रोतांस्याविश्य कुप्यति।
उरःस्थः कफमुद्धूय हिक्काश्वासान् करोति सः॥
घोरान् प्राणोपरोधाय प्राणिनां पञ्च पञ्च च।
C.S.Ci. 17/17-18

यदा स्त्रोतांसि संरुध्य मारुतः कफपूर्वकः।
विष्वग्व्रजति संरुद्धस्तदा श्वासान्करोति सः॥
C.S.Ci. 17/45

Due to consumption of etological factors *Prāṇavāta* combining with *Kapha* leaves its own path and begins to move in the upward direction, giving rise to more of respirations is known as *Śvāsa Roga & Hikkā Roga,* which are of five kinds each & which may lead to the death of the patient.

कफवातात्मकावेतौ पित्तस्थानसमुद्भवौ।
हृदयस्य रसादीनां धातूनां चोपशोषणौ॥
तस्मात् साधारणावेतौ मतौ परमदुर्जयौ।
मिथ्योपचरितौ क्रुद्धौ हत आशीविषाविव॥
C.S.Ci. 17/8-9

Both *Hikkā & Śvāsa* originates from the seat of *Pitta* and are caused by the simultaneous aggravation of *Kapha & Vāta* and dries up the *Hṛdaya & Rasādi Dhātus.* Hence both being similar are regarded as very difficult to treat and if treated inappropriately leading to further aggravation of the condition & kill the patient like serpents.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphadi doṣās*
↓
Development of *Āma* inside the gastro intestinal tract and *Śvāsa* originates from the seat of *Pitta*
↓
Localisation of *Kapha doṣa* (especially *Rasadhātugata Malarupi Kapha)* in the *Prāṇavaha Srotas*
↓
Leading to aggravation of *Vāta doṣa* inside the *Prāṇavaha Srotas* leaves its own path and begins to move in the upward direction
↓
Dries up the *Hṛdaya & Rasādi Dhātus.*
↓
Śvāsa Roga

श्वास रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Śvāsa Roga)

Doṣa : *Vāta-Kapha* (*Prāṇa* and *Udāna Vāta, Bhodaka & Kledaka Kapha*)
Dūṣya : *Dhātu → Rasa Dhātu*
Upadhātu →
Śārīrika mala → Mūtra, Puriṣa
Dhātumala→Kapha, Pitta, Tvacā and *Sveda*
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Prāṇavaha, Udakavaha, Annavaha and Rasavaha*
Srotoduṣṭi : *Saṅga & Vimārgamana*
Udbhavasthāna : *Āmāśayottha (Pittasthāna)*
Vyaktasthāna : *Āsya, Grīvā, Pṛṣṭa, Pārśva*
Sancārasthāna : *Prāṇavaha Sroto Avayava*
Adhiṣṭhāna : *Pitta Sthāna*
Prabhāva : *Kṣudra Śvāsa→Sādhya*
Tamaka Śvāsa→ Kṛcchra Sādhya Mahā, Cinna and Urdvaśvāsa→ Asādhya
Svabhāva : *Āśukāri*
Rogamārga : *Madhyama Roga Mārga*
Roga Bheda : 5 kinds

पूर्वरूप (Pūrvarupa)

प्राग्रूपं तस्य हृत्पीडा भक्तद्वेषोऽरतिः परा।
आनाहः पार्श्वयोः शूलं वैरस्यं वदनस्य च॥
S.S.Utt. 51/6

आनाहः पार्श्वशूलं च पीडनं हृदयस्य च।
प्राणस्य च विलोमत्वं श्वासानां पूर्वलक्षणम्॥
C.S.Ci. 17/20

Premonitory signs & symptoms of Śvāsa are as follows:- Pain in chest; aversion towards food; restlessness; flatulence; pain in the flanks; bad taste in the mouth and reversion of respiratory functions.

रोग भेद (Roga Bheda)

क्षुद्रकस्तमकश्छिन्नो महानूर्द्ध्वश्च पञ्चधा।
भिद्यते स महाव्याधिः श्वास एको विशेषतः॥
S.S.Utt. 51/5

Śvāsa is difficult to tolerate, incurable & dreadful disease and it is only one variety. But it is classified further into five kinds for easy understanding of the disease namely *Kṣudra, Tamaka, Chinna, Mahā & Ūrdhva.*

महाश्वास (Mahāśvāsa)

उद्धूयमानवातो यः शब्दवद्दुःखितो नरः।
उच्चैः श्वसिति संरुद्धो मत्तर्षभ इवानिशम्॥
प्रनष्टज्ञानविज्ञानस्तथा विभ्रान्तलोचनः।
विकृताक्ष्याननो बद्धमूत्रवर्चा विशीर्णवाक्॥
दीनः प्रश्वसितं चास्य दूराद्विज्ञायते भृशम्।
महाश्वासोपसृष्टः स क्षिप्रमेव विपद्यते॥
इति महाश्वासः।
C.S.Ci. 17/46-48

निःसंज्ञः पार्श्वशूलार्त्तः शुष्ककण्ठोऽतिघोषवान्।
संरब्धनेत्रस्त्वायम्य यः श्वस्यात् स महान् स्मृतः॥
S.S.Utt. 51/12

Mahāsvāsa is characterised by deep breath associated with discomfort and sound like that of mad bull or intoxicated bull due to obstruction in the *Prāṇavaha Srotas.* Because of this, *Vāta* moves forcibly in upward direction. Patient loosses all sorts of knowledge, rolling eyes, distortion of eyes & face, retention of urine & stool, feeble voice, anxious expression, respiration sound becomes audible even from a distance. It is known as *Mahāsvāsa* & succumbs to death instantaneously. Patient may become unconscious, pain in the flanks, dryness of throat, swollen eyes & stretching of body during breathing.

ऊर्ध्वश्वास (Ūdhvaśvāsa)

दीर्घं श्वसिति यस्तूर्ध्वं न च प्रत्याहरत्यधः।
श्लेष्मावृतमुखस्त्रोताः क्रुद्धगन्धवहार्दितः॥
ऊर्ध्वदृष्टिर्विपश्यंश्च विभ्रान्ताक्ष इतस्ततः।
प्रमुह्यन् वेदनार्तश्च शुष्कास्योऽरतिपीडितः॥
ऊर्ध्वश्वासे प्रकुपिते ह्यधःश्वासो निरुध्यते।
मुह्यतस्ताम्यतश्चोर्ध्वं श्वासस्तस्यैव हन्त्यसून्॥
इत्यूर्ध्वश्वासः। *C.S.Ci. 17/49-51*

मर्मस्वायम्यमानेषु श्वसन्मूढो मुहुश्च यः।
ऊद्‌र्ध्वप्रेक्षी हतरवस्तमूद्‌र्ध्वश्वासमादिशेत्॥
S.S.Utt. 51/13

Ūrdhvaśvāsa is characterised by forceful expiration and impeded inspiration because of which the patient becomes unconscious with tremors and finally dies. It is associated with prolonged expiration & inability to have inspiration, adhesion of sputum in mouth & *Prāṇavaha Srotas,* affliction with aggravated *Vāta,* gazes abnormally with eyes up & rolling, unconsciousness, suffering from severe pain, dryness of mouth, restlessness, patient feels stretching kind of pain in vital parts (*Marmās*), faints oftenly after respiration and loss of voice.

छिन्नश्वास (Chinnaśvāsa)

यस्तु श्वसिति विच्छिन्नं सर्वप्राणेन पिडितः।
न वा श्वसिति दुःखार्तो मर्मच्छेदरुगर्दितः॥
आनाहस्वेदमूर्च्छार्तो दह्यमानेन बस्तिना।
विप्लुताक्षः परिक्षीणः श्वसन् रक्तैकलोचनः॥
विचेताः परिशुष्कास्यो विवर्णः प्रलपन्नरः।
छिन्नश्वासेन विच्छिन्नः स शीघ्रं प्रजहात्यसून्॥
इति छिन्नश्वासः। *C.S.Ci.18/52-54*
आध्मातो दह्यमानेन बस्तिना सरुजं नरः।
सर्वप्राणेन विच्छिन्नं श्वस्याच्छिन्नं तमादिशेत्॥
S.S.Utt. 51/11

Chinnaśvāsa is characterised by intermittent respiration or no respiration at all or painful respiration due to affliction of all the channels carrying *Prāṇavāta;* cutting pain in vital parts; *Ānāha;* perspiration; fainting; burning sensation in urinary bladder; tearing eyes; extreme emaciation; redness in one eye; detraction of mind; dryness in the mouth; abnormal complexion; delirium. Patient succumbs to death instantaneously due to cut off by the intermittent respiration.

तमकश्वास (Tamaka Śvāsa)

प्रतिलोमं यदा वायुः स्त्रोतांसि प्रतिपद्यते।
ग्रीवां शिरश्च संगृह्य श्लेष्माणं समुदीर्य च॥
करोति पीनसं तेन रुद्धो घुर्घुरुकं तथा।
अतीव तीव्रवेगं च श्वासं प्राणप्रपीडकम्॥
प्रताम्यत्यतिवेगाच्च कासते सन्निरुध्यते।
प्रमोहं कासमानश्च स गच्छति मुहुर्मुहुः॥
श्लेष्मण्यमुच्यमाने तु भृशं भवति दुःखितः।
तस्यैव च विमोक्षान्ते मुहूर्तं लभते सुखम्॥
अथास्योद्ध्वंसते कण्ठः कृच्छ्राच्छक्नोति भाषितुम्।
न चापि निद्रां लभते शयानः श्वासपीडितः॥
पार्श्वे तस्यावगृह्णाति शयानस्य समीरणः।
आसीनो लभते सौख्यमुष्णं चैवाभिनन्दति॥
उच्छ्रिताक्षो ललाटेन स्विद्यता भृशमर्तिमान्।
विशुष्कास्यो मुहुः श्वासो मुहुश्चैवावधम्यते॥
मेघाम्बुशीतप्राग्वातैः श्लेष्मलैश्चाभिवर्धते।
स याप्यस्तमकश्वासः साध्यो वा स्यान्नवोत्थितः॥
इति तमकश्वासः। *C.S.Ci.17/55-62*

तृटस्वेदवमथुप्रायः कण्ठघुर्घुरिकान्वितः।
विशेषाद् दुर्दिने ताम्येच्छवासः स तमको मतः॥
घोषेण महताऽऽविष्टः सकासः सकफो नरः।
यः श्वसित्यबलोऽन्नद्विट् सुप्तस्तमकपीडितः॥
S.S.Utt. 51/8-9

Due to obstruction in *Prāṇavaha Srotas, Vāta* moves in reverse order and pervades the entire *Prāṇavaha Srotas* and stimulates the *Kapha* in the throat & head causing rhinitis. Further it manifest wheezing sound; greater enhancement of respiration in relation to rate & rhythm; feeling of darkness in front of the eyes; thirst; vomiting; paroxysmal attack of this disease are severe enough to put the patient in distress, to bend him forward, to cough again & again which sometimes result in fainting; until the expectoration of provoked *Kapha* patient remains agony and patient finds temporary relief; after expectorating *Kapha* patient is unable to talk much because of choking in the throat; aversion to food; does not get sleep when he lies down because of catching of breath; finds comfort in warm substances; eyes wide opened; sweating in forehead; restlessness; dryness of the mouth; suffers frequently from paroxysms of dyspnoea which aggravates during cloudy days, by the use of cold water, breeze from east and consumption of *Kapha* aggravating factors. It is manageable in chronic stage and curable if it is of recent onset.

प्रतमक श्वास (Pratamaka Śvāsa) & संतमक श्वास (Santamaka Śvāsa)

स शाम्यति कफे हीने स्वपतश्च विवर्द्धते।
मूर्च्छाज्वराभिभूतस्य ज्ञेयः प्रतमकस्तु सः॥
S.S.Utt. 51/10

ज्वरमूर्च्छापरीतस्य विद्यात् प्रतमकं तु तम्।
उदावर्तरजोऽजीर्णक्लिन्नकायनिरोधजः॥
तमसा वर्धतेऽत्यर्थं शीतैश्चाशु प्रशाम्यति।
मज्जतस्तमसीवाऽस्य विद्यात् संतमकं तु तम्॥
इति प्रतमकसंतमकश्वासौ। *C.S.Ci. 10/63-64*

Disease which subsides when *Kapha* decreases & increases greatly when sleeping and associated with fainting & fever is known as *Pratamaka*. It is caused by *Udāvarta,* dust, indigestion, humidity, suppression of natural urges by an old person. It manifest due to aggravation of *Pitta.*

Santamaka Śvāsa manifest due to increase of *Tamas* and gets relieved quickly by cooling regimes. These patients feels as if they submerged in darkness.

क्षुद्र श्वास (Kṣudra Śvāsa)

रुक्षायासोद्भवः कोष्ठे क्षुद्रो वात उदीरयन्।
क्षुद्रश्वासो न सोऽत्यर्थं दुःखेनाङ्गप्रबाधकः॥
हिनस्ति न स गात्राणि न च दुःखो यथेतरे।
न च भोजनपानानां निरुणद्ध्युचितां गतिम्॥
नेन्द्रियाणां व्यथां नापि कांचिदापादयेद्रुजम्।
स साध्य उक्तो बलिनः सर्वे चाव्यक्तलक्षणाः॥
C.S.Ci. 517/65-67

किञ्चिदारभमाणस्य यस्य श्वासः प्रवर्त्तते।

निषण्णस्यैति शान्तिञ्च स क्षुद्र इति संज्ञितः ॥

S.S.Utt. 51/7

Kṣudra Śvāsa manifest due to mild aggravation of *Vāta* in the *Koṣṭha* on account of physical exertion and rough regimens. It subsides when person takes rest. It does not produce discomfort, does not afflict body parts, it is not troublesome as other forms of *Śvāsa* are, does not restrict the normal movement of foods & drinks, does not produce any discomfort or pain in sense organs. It is curable.

Prognosis

All other forms of *Śvāsa* are curable when they are mild & affecting strong persons.

साध्यासाध्यता (Sādhyāsādhyatā)

एषां प्राणहरा वर्ज्या घोरास्ते ह्याशुकारिणः ॥
भेषजैः साध्ययाप्यांस्तु क्षिप्रं भिषगुपाचरेत् ।
उपेक्षिता दहेयुर्हि शुष्कं वृक्षमिवानलः ॥

C.S.Ci. 17/68-69

Incurable varieties of *Śvāsa* are fatal because they are very severe & galloping. That's why these kinds of *Śvāsa* should not be treated.

On the contrary curable & manageable varieties of *Śvāsa* should be treated with proper medicaments because if neglected or delayed they may reduce the body to ashes like the fire burning the dried tree.

क्षुद्रः साध्यतमस्तेषां तमकः कृच्छ्र उच्यते ।
त्रयः श्वासा न सिध्यन्ति तमको दुर्बलस्य च ॥

S.S.Utt. 51/14

Kṣudra Śvāsa is curable easily, *tamaka Śvāsa* is difficult to cure, *Ūrdhva, Maha, Chinna Śvāsa* in weak patients is incurable.

उपशय/पथ्य (Upaśaya/Pathya)

The useful items in the case of dysponea includes the following: Sweating, smoking herbal drugs, sleep during daytime, intake of such items as the red variety of rice, *Kulattha* (dolichos bean), wheat, barley, flesh of rabit, peacock, partridge, wood-cutter, chicken, parrot and the like birds and animals of the arid regions, old *Ghṛta*, milk and *Ghṛta* of the goat, liquor, honey, leaves of *Nidigdhikā*, *Vāstuka* (chenopodium), *Taṇḍulīya*, *Jīvantī*, young plant of the radish, *Paṭola*, brinjal, garlic, *Harada*, *Jambīra* lemon, *Bimbī* fruits, *Mātuluṅga* lemon, date fruits, smaller cardamom, *Puṣkaramūla*, hot water, *Trikaṭu*, urine of the cow and all other items that reduces the *Kapha* and *Vāta* contents of the body. Also helpful to the patient of *Śvāsa* is the cauterization on both sides of his breast, on the middle fingers of his hands as well as around the pit of his throat. (B.R. 16/132-136)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Harmful activities and items in cases of Śvāsa roga includes : Suppression of the natural urges, nasal therapies, the *Vasti* therapy, chewing wood sticks with the teeth, hard labour, excessive walking, lifting heavy items, facing dust, moving out in sun, consuming meals heavy to digest, having intercourse, taking hot items of meal, taking meat of the animals from the marshy region, taking deep fried nuggets and the like dishes, taking items that produce *Kapha*, taking the *Urada* beans, blood-letting, facing the easternly wind, drinking water immediately after eating meals, taking the milk and *Ghṛta* of sheep, drinking impure water, eating fishes, vegetable leaves of *Kanda* and the like tubers, leaves of mustard, etc. (B.R. 16/137-139).

MCQs

1. *Amapradosha anaha roukshyadanyapatrpanat* is the Nidana of
   a. *Shwasa*
   b. *Gulma*
   c. *Grahani*
   d. *Pravahika*
2. *Rakthaikalocana* is seen in
   a. *Chinna Shwasaa*
   b. *Mahashwasa*
   c. *Urdhwashwasa*
   d. *Tamakashwasa*
3. *Dosha* responsible for *Shwasa* is
   a. *Prana-Udana*
   b. *Udana-Vyanaa*
   c. *Prana-Kapha*
   d. *Apana-Prana*
4. *Viplutaksha parikshina mentioned in context to*
   a. *Chinna Shwasa*
   b. *Mahashwasa*
   c. *Urdhwashwasa*
   d. *Tamakashwasa*

5. *Pranastajnana vijnana statha vibhranta lochana* is the feature of

   a. *Chinna Shwasa* b. *Mahashwasa*
   c. *Urdhwashwasa* d. *Tamakashwasa*

6. *Mattarshabha ivanisham* is the feature of

   a.*Tamakashwasa* b. *Mahashwasa*
   c. *Urdhwashwasa* d. *Chinna Shwasa*

7. *Deenaha prashwasitam chasya duradvijnayate bhrusham observed in*

   a. *Chinna Shwasaa* b. *Urdhwashwasa*
   c. *Mahashwasa* d. *Tamakashwasa*

8. *Srotas* involved in the *Samprapti* of *Swasa*

   a. *Pranavaha* b. *Mutravaha*
   c. *Udaka vaha* d. *Anna vaha*

9. *Anaha sweda murcharto dahyamanena bastina* is the feature of

   a. *Chinna Shwasaa* b. *Mahashwasa*
   c. *Urdhwashwasa* d. *Mutrakricchra*

10. *Sa shamyati kaphe hine swapatascha vivardhate feature observed in*

    a. *Pratamaka swasha*
    b. *Santamaka swasha*
    c. *Khudra swasha*
    d. *Tamakashwasha*

11. *Tritswedavamathu prayahkanthaghurghurikanvitah* feature observed in

    a. *Pratamaka swasha*
    b. *Santamaka swasha*
    c. *Khudra swasha*
    d. *Tamakashwasha*

Answer

1.	a	2.	a	3.	c
4.	a	5.	a	6.	b
7.	c	8.	a	9.	a
10.	a	11.	d		

CHAPTER 14

Kasa Roga

Introduction

Kāsa is one of the diseases of *Prāṇvaha srotas.* The disease *Kāsa* has been named on the basis of its *Pratyātma lakṣaṇā* (cardinal symptom). It may occur as a separate disease or as a symptom in other diseases. *Acārya Caraka* and *Suśruta* explained *Kāsa* after *Hikkā* and *Śvāsa* because of their similarity in *Nidāna* and *Cikitsā.* But *Vāgbhaṭa* explained *Kāsa* along with *Raktapitta* due to the acuteness of its manifestation and also *Raktapitta* (haemoptysis) is a complication of an untreated *Kāsa.* A cough is an action the body takes to get rid of substances that are irritating to the air passages, which carry the air a person breathes in from the nose and mouth to the lungs.

A cough occurs when cells along the air passages get irritated and trigger a chain of events. The result is air in the lungs is forced out under high pressure. A person can choose to cough (a voluntary process), or the body may cough on its own (an involuntary process).

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

कसृ शब्द कुत्सायाम् । *Amarkoṣa*
कासः कुत्सित शब्दः ॥ *Śabdakalpdruma*
कासः कुत्सित शब्दः ॥ *Vācaspatyam*

The term *Kāsa* is derived from the root *'Kasṛ'* meaning *"hard"* which refer to *Kutsāyām* (contempt). Therefore *Kāsa* factually means the air in the *Prāṇvaha srotas* is forced out under high pressure making unpleasant or contempt sound.

कासतेऽनेन कासः । *Vācaspatyam*
कासनं कास इति ॥ *M.Ni. 11/1-2 (Madhu)*
कसनं कासः ॥ *Āḍd.Śa.Prā. 7/21*

It is known as *Kāsa* due to occurrence of symptom *Kāsa* (cough)

कसति शिरः कण्ठात् ऊर्ध्वं गच्छति वायुरिति कासः ॥
"कस" गतौ इत्यस्मात्; कसनात् कासः ॥
M.Ni. 11/1-2 (Madhu)

The word *Kāsa* is derived from *dhātu "Kasa"* which refers to carry the air a person breathes in from the organs of *Prāṇvaha srotas* refers to *Kāsa* (cough). Pathologically, in the disease *Kāsa,* the vitiated *Vāta* forcibly comes out with upward movement through the channels situated in the head and throat by producing a peculiar sound.

पर्याय (Paryāya)

कासस्तु क्षवथुः ॥ *Amar. Manuṣyavarg. 352*
कासः क्षवथुरुच्यते ॥ *Rā.Ni.Rogādivarg. 20/3*

In *Amarakoṣa* and *Rāja nighaṇṭu Kṣavathu* is mentioned as the synonym of *Kāsa.*

हेतु (Hetu)

उक्ता ये हेतवो नृणां रोगयोः श्वासहिक्कयोः ।
कासस्यापि च विज्ञेयास्त एवोत्पत्तिहेतवः ॥
धूमोपघाताद्रजसस्तथैव व्यायामरूक्षान्ननिषेवणाच्च ।
विमार्गगत्वादपि भोजनस्य वेगावरोधात् क्षवथोस्तथैव ॥
S.S.Utt. 52/3-4

Factors which are responsible for the manifestation of *Hikkā* & *Śvāsa* are also responsible for the development of *Kāsa.*

Exposure to smoke & dust; excessive physical exercises; consumption of rough foods; entry of food substances in abnormal path & suppression of sneezing.

कास रोग संप्राप्ति (Samprāpti of Kāsa Roga)

अधःप्रतिहतो वायुरुर्ध्वस्त्रोतःसमाश्रितः ।
उदानभावमापन्नः कण्ठे सक्तस्तथोरसि ॥
आविश्य शिरसः खानि सर्वाणि प्रतिपूरयन् ।
आभञ्जन्नाक्षिपन् देहं हनुमन्ये तथाऽक्षिणी ॥
नेत्रे पृष्ठमुरःपार्श्वे निर्भुज्य स्तम्भयंस्ततः ।
शुष्को वा सकफो वाऽपि कसनात्कास उच्यते ॥
प्रतिघातविशेषेण तस्य वायोः सरंहसः ।
वेदनाशब्दवैशिष्ट्यं कासानामुपजायते ॥
C.S.Ci. 18/6-9

प्राणो ह्युदानानुगतः प्रदुष्टः सम्भिन्नकांस्यस्वनतुल्यघोषः ।
निरेति वक्त्रात्सहसा सदोषः कासः स विद्वद्भिरुदाहृतस्तु ॥
S.S.Utt. 52/5

Due to above etiological factors *Kapha* gets aggravated and brings obstruction for the movement of *Vāta* in the *Uras* leading to aggravation of *Prāṇavāta* and *Udāna Vāta*. As a result *Vāta* moves upwards, afflicts the channels of circulation in the upper part of the body and gets lodged in the throat & chest. This agitated *Vāta* enters & fills up all the channels of *Śiras* to cause bending & stretching of the body, jaws, sides of neck & eyes. Further *Vāta* brings contraction & stiffness of the eyes, back, chest & sides of the chest, gives rise to *Kāsa* which may be dry or with sputum. It is associated with severe pain & loud sound like that of a broken bronze.

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kaphādi doṣās*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Localisation of *Kapha doṣa* (especially *Rasadhātugata Malarupi Kapha*) in the *Prāṇavaha Srotas*

↓

Leading to aggravation of *Vāta doṣa* inside the *Prāṇavaha Srotas* (mainly throat & chest) which moves upwards

↓

The agitated *Vāta* enters & fills up all the channels of *Śiras* to cause bending & stretching of the body, jaws, sides of neck & eye along with it also brings contraction & stiffness of the eyes, back, chest & sides of the chest

↓

Kāsa

↓

Which may be dry or with sputum and it is associated with severe pain & loud sound like that of a broken bronze.

कास रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Kāsa Roga)

Doṣa : *Vāta Kapha Pradhāna Tridoṣaja*
Vāta→ Prāna, Udāna & Apāna Vāta
Pitta→ Pācaka
Kapha→ Kledaka & Avalambaka
Dūṣya : *Dhātu → Rasa Dhātu*
Upadhātu → Śirā
Śārīrika Mala
Dhātu Mala → Kapha
Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Rasavaha Srotas*
Prāṇavaha Srotas, Annavaha Srotas
Srotoduṣṭi : *Saṅga & Vimārgamana*
Udbhavasthāna : *Āmāśayottha; Antaḥmārga (Koṣṭha-Amāśaya C.Su 11/17)*
Sancārasthāna : *Prāṇavaha Sroto Avayava*
Vyaktasthāna : *Mukha, Śira, Pṛṣṭa, Pārśva (Caraka)*
Hṛdaya, Śankha, Mūrdha (Suśruta)
Adhiṣṭhāna : *Uras (Prāṇavahasrotas)*
Svabhāva : *Āśukāri → Dhūmopaghātajanya, Bhojanasya Vimārgajanya Cirakāri*
Prabhāva : *Vātaja, Pittaja & Kaphaja → Sādhya*
Kṣataja Kāsa & Kṣayaja Kāsa→Yāpya

कास भेद (Kāsa Bheda)

स वातपित्तप्रभवः कफाच्च क्षतात्तथाऽन्यः क्षयजोपरश्च ।
पञ्चप्रकारः कथितो भिषग्भिर्विवर्द्धितो यक्ष्मविकारकृत् स्यात् ॥
S.S.Utt. 52/6

वातादिजास्त्रयो ये च क्षतजः क्षयजस्तथा ।
पञ्चैते स्युर्नृणां कासा वर्धमानाः क्षयप्रदाः ।।
C.S.Ci. 18/4

Kāsa is classified into five varieties namely :

1) *Vātaja*
2) *Pittaja*
3) *Kaphaja*,
4) *Kṣataja*
5) *Kṣayaja*.

पूर्वरूप (Pūrvarupa)

पूर्वरुपं भवेत्तेषां शूकपूर्णगलास्यता ।
कण्ठे कण्डूश्च भोज्यानामवरोधश्च जायते ॥

C.S.Ci. 18/5

भविष्यतस्तस्य तु कण्ठकण्डूर्भोज्योपरोधो गलतालुलेपः ।
स्वशब्दवैषम्यमरोचकोऽग्निसादश्च लिङ्गानि भवन्त्यमूनि ॥

S.S.Utt. 52/7

Premonitory signs & symptoms of Kāsa are as follows : Patient feels that throat and mouth are filled with a feeling of congestion in the throat; itching sensation in the throat; hinderance to swallowing; coating of throat & palate; hoarseness of voice; loss of taste and mildness of digestive fire.

वातज कास (Vātaja Kāsa)

वातज कास निदान (Nidāna of Vātaja Kāsa)

रुक्षशीतकषायाल्पप्रमितानशनं स्त्रियः ।
वेगधारणमायासो वातकासप्रवर्तकाः ॥

C.S.Ci. 18/10

Vātaja Kāsa manifest due to consumption of the following etiological factors:- Intake of rough, cold & astringent food; consumption of food in less quantity; inadequate intake of food; fasting; excessive sexual intercourse; suppression of natural urges and excessive physical strain.

वातज कास लक्षण (Lakṣaṇa of Vātaja Kāsa)

हृत्पार्श्वोरःशिरःशूलस्वरभेदकरो भृशम् ।
शुष्कोरःकण्ठवक्त्रस्य हृष्टलोम्नः प्रताम्यतः ॥
निर्घोषदैन्यस्तननदौर्बल्यक्षोभमोहकृत् ।
शुष्ककासः कफं शुष्कं कृच्छ्रान्मुक्त्वाऽल्पतां व्रजेत् ॥
स्निग्धाम्ललवणोष्णैश्च भुक्तपीतैः प्रशाम्यति ।
ऊर्ध्ववातस्य जीर्णेऽन्ने वेगवान्मारुतो भवेत् ॥

C.S.Ci. 18/11-13

हृच्छङ्खमूर्धोदरपार्श्वशूली क्षामाननः क्षीणबलस्वरौजाः।
प्रसक्तमन्तः कफमीरणेन कासेत्तु शुष्कं स्वरभेदयुक्तः ॥

S.S.Utt. 52/8

Excruciating pain in the cardiac region, sides of the chest, abdomen, temples, chest & head; hoarseness of voice; dryness of chest, throat & mouth; horripilation, feeling of darkness, sounds like that of conch or clouds during coughing; anxious expression; debility; irritation & fainting; dry cough; patient feels difficulty in expectoration of sputum because of less quantity & dryness of sputum; symptoms gets relieved by foods & drinks which are unctous, sour, saline & warm. Symptoms gets aggravated during empty stomach and diminution of strength, voice & *Ojas.*

पित्तज कास (Pittaja Kāsa)

पित्तज कास निदान (Nidāna of Pittaja Kāsa)

कटुकोष्णविदाह्यम्लक्षारणामतिसेवनम् ।
पित्तकासहरं क्रोधः संतापश्चाग्निसूर्यजः ॥

C.S.Ci. 18/14

Pittaja Kāsa manifest due to following etiological factors : Excessive consumption of pungent, hot, *Vidāhi,* sour & alkaline food, anger; exposure to the heat of fire & sun.

पित्तज कास लक्षण (Lakṣaṇa of Pittaja Kāsa)

पीतनिष्ठीनाक्षित्वं तिक्तास्यत्वं स्वरामयः ।
उरोधूमायनं तृष्णा दाहो मोहोऽरुचिर्भ्रमः ॥
प्रततं कासमानश्च ज्योतींषीव च पश्यति ।
श्लेष्माणं पित्तसंसृष्टं निष्ठीवति च पैत्तिके ॥

C.S.Ci. 18/15-16

उरोविदाहज्वरवक्त्रशोषैरभ्यर्दितस्तिक्तमुखस्तृषार्त्तः ।
पित्तेन पीतानि वमेत्कटूनि कासेत्सपाण्डुः परिदह्यमानः ॥

S.S.Utt. 52/9

Signs & symptoms of Pittaja Kāsa are as follows : Yellowishness of sputum & eyes; bitterness in the mouth; disorders of voice; patient feels as if smoke is coming out of *Uras;* thirst; burning sensation; fainting; anorexia & giddiness; due to continous cough patient feels appearance of star particles infront of the eyes; patient expectorates sputum mixed with *Pitta;* fever; dryness of mouth; vomiting of yellow & pungent materials and cough accompanied with burning sensation in the throat.

कफज कास (Kaphaja Kāsa)

कफज कास निदान (Nidāna of Kaphaja Kāsa)

गुर्वभिष्यन्दिमधुरस्निग्धस्वप्नाविचेष्टनैः ।
वृद्धः श्लेष्माऽनिलं रुद्ध्वा कफकासं करोति हि ॥

C.S.Ci. 18/17

Kaphaja Kāsa manifest due to following etiological factors: Consumption of heavy; *Abhiṣyandi* (ingredients which cause obstruction to channels); sweet and unctous substances; excessive sleep and sedentary lifestyle. Due to above etiological factors *Kapha* gets aggravated and obstructs the movement of *Vāta* leading to manifestation of *Kaphaja Kāsa.*

कफज कास लक्षण (Lakṣaṇa of Kaphaja Kāsa)

मन्दाग्नित्वारुचिच्छर्दिपीनसोत्क्लेशगौरवैः ।
लोमहर्षास्यमाधुर्यक्लेदसंसदनैर्युतम् ॥
बहुलं मधुरं स्निग्धं निष्ठीवति घनं कफम् ।
कासमानो ह्यरुग् वक्षः संपूर्णमिव मन्यते ॥

C.S.Ci. 18/18-19

प्रलिप्यमानेन मुखेन सीदन् शिरोरुजार्त्तः कफपूर्णदेहः ।
अभक्तरुग्गौरवसादयुक्तः कासेत ना सान्द्रकफं कफेन ॥

S.S.Utt. 52/10

Mildness of digestive fire; anorexia; vomiting; chronic rhinitis; nausea; heavyness of the body; horripilation; sweetness & stickiness of mouth; general malaise; chest is full of sputum and expectorates profuse, sweet, unctous & viscid sputum without pain.

क्षतज कास (Kṣataja Kāsa)

क्षतज कास निदान (Nidāna of Kṣataja Kāsa)

अतिव्यवायभाराध्वयुद्धाश्वगजविग्रहैः ।
रुक्षस्योरः क्षतं वायुर्गृहीत्वा कासमावहेत् ॥

C.S.Ci. 18/20

वक्षोऽतिमात्रं विहतं तु यस्य व्यायामभाराध्ययनाभिघातैः ।
विश्लिष्टवक्षाः स नरः सरक्तं ष्ठीवत्यभीक्ष्णं क्षतजं तमाहुः ॥

S.S.Utt. 52/11

Kṣataja Kāsa manifest by following etiological factors:- Excessive indulgence in sex; weight lifting; walking long distances; indulgence in fighting; combat with horses & elephants; studying in loud voice and injuries.

Due to above etiological factors chest becomes injured and bring about roughness in the body leading to aggravation of *Vāta* which gives rise to *Kṣataja Kāsa.*

क्षतज कास लक्षण (Lakṣaṇa of Kṣataja Kāsa)

स पूर्वं कासते शुष्कं ततः ष्ठीवेत् सशोणितम् ।
कण्ठेन रुजताऽत्यर्थं विरुग्णेनेव चोरसा ॥
सूचीभिरिव तीक्ष्णाभिस्तुद्यमानेन शूलिना ।
दुःखस्पर्शेन शूलेन भेदपीडाभितापिना ॥
पर्वभेदज्वरश्वासतृष्णावैस्वर्यपीडितः ।
पारावत इवाकूजन् कासवेगात्क्षतोद्भवात् ॥

C.S.Ci. 18/21-23

Signs & symptoms of Kṣataja Kāsa are as follows:- Patient suffers from dry cough followed by expectoration of bloody sputum; intense pain in the throat; excruciating, piercing and breaking pain with tenderness in chest; pain in small joints; fever; dyspnoea; thirst; hoarseness of voice; while coughing patient produces cooing sound like that of a pigeon.

क्षयज कास (Kṣayaja Kāsa)

क्षयज कास निदान (Nidāna of Kṣayaja Kāsa)

विषमासात्म्यभोज्यातिव्यवायाद्वेगनिग्रहात् ।
घृणिनां शोचतां नृणां व्यापन्नेऽग्नौ त्रयो मलाः ॥
कुपिताः क्षयजं कासं कुर्युर्देहक्षयप्रदम् ।

C.S.Ci. 18/24-25

Kṣayaja Kāsa manifest due to following etiological factors:- Consumption of irregular meals; unwholesome food; excessive indulgence in sex; suppression of natural urges; immensely hateful disposition and excessive worry. Due to above etiological factors *Agni* becomes deranged along with aggravation of all the three *Doṣās* leading to manifestation of *Kṣayaja Kāsa,* which further causes emaciation of the body.

क्षयज कास लक्षण (Lakṣaṇa of Kṣayaja Kāsa)

दुर्गन्धं हरितं रक्तं ष्ठीवेत् पूयोपमं कफम् ॥
स्थानादुत्कासमानश्च हृदयं मन्यते च्युतम् ।
अकस्मादुष्णशीतार्तो बह्वाशी दुर्बलः कृशः ॥
स्निग्धाच्छमुखवर्णत्वक् श्रीमद्दर्शनलोचनः ।
पाणिपादतलैः श्लक्ष्णैः सततासूयको घृणी ॥
ज्वरो मिश्राकृतिस्तस्य पार्श्वरुक् पीनसोऽरुचिः ।
भिन्नसंहतवर्चस्त्वं स्वरभेदोऽनिमित्ततः ॥
इत्येष क्षयजः कासः क्षीणानां देहनाशनः ।

C.S.Ci. 18/25-29

Signs & symptoms of Kṣayaja Kāsa are as follows:- Patient expectorates sputum which is foul smelling, green or red in colour and appears like pus; patient feels displacement of heart from its normal place; patient suddenly feels heat and again cold; excess consumption of food but patient feels weak & emaciated; face, complexion and skin becomes clean & unctous; patient appearance become gracious with his eyes glowing; sole & feet become smooth; envious; disgustful; suffers from fever having mixed characters; pain in sides; chronic rhinitis anorexia; liquid or solid stool and hoarseness of voice without an apparent cause; burning sensation; delusion; decrease of respiration (*Prāṇakṣaya*); depletion of *Māmsa Dhātu* and kills the emaciated patient.

साध्यासाध्यता (Sādhyāsādhyatā)

इत्येष क्षयजः कासः क्षीणानां देहनाशनः।
साध्यो वलवतां वा स्याद्याप्यस्त्वेवं क्षतोत्थितः॥
नवौ कदाचित् सिध्येतामेतौ पादगुणान्वितौ।
स्थविराणां जराकासः सर्वो याप्यः प्रकीर्तितः॥
C.S.Ci. 18/2 9-30

तं सर्वलिङ्गं भृशदुश्चिकित्स्यं चिकित्सितज्ञाः क्षयजं वदन्ति।
वृद्धत्वमासाद्य भवेत्तु यो वै याप्यं तमाहुर्भिषजस्तु कासम्॥
S.S.Utt. 52/13

Kṣayaja Kāsa is curable in strong persons. If all the signs & symptoms present then it becomes difficult for treatment. Advanced stage of *Kṣayaja Kāsa* is incurable.

Kṣataja Kāsa is palliable if the patient is strong.

If *Kṣataja Kāsa* & *Kṣayaja Kāsa* are in their initial stage & if all the four limbs of treatment are excellent then these two are curable at times.

All varieties of *Kāsa* are manageable in old age people.

उपशय/पथ्य (Upaśaya/Pathya)

Diet and drinks for *Vātika Kāsa*: *Vāstuka*, *Vāyas*, *Mūlaka*, *Suniṣaṇṇaka*, unctuous substance like oil, milk, sugar cane juice, preparations of *Guḍa*, curd, *Aranāla* and sour fruits, *Prasannā* type of alcoholic drink and others which are either sweet, sour or saline in taste are useful for a patient having *Vātika* type of *Kāsa*. These items should be used along with soup of the meat of domesticated, marshy and aquatic animals. (B.R. 15/1-3)

Intake of the soup of the meat of animals dwelling in arid land, sweet eatables, *Śyāmaka*, *Yava*, *Kodravā*, soup of *Mudga*, etc. and vegetables having bitter taste in appropriate quantity is useful for a patient suffering from *Paittika* type of *Kāsa*. (B.R. 15/8)

The following are helpful measures for the patient of *Kāsa:- Svedana* therapy, *Virecana* (purgation) therapy, vomiting, smoke therapy, taking meals on right time and in limited quantity. He should also take the following items in his meals: separate juices of *Śālidhānya* rice, *Ṣāṣṭhi* rice, wheat, *Śyāmāka*, *Yava*, *Kodarva*, seeds of *Ātmaguptā*, *Urada*, *Mudga* and *Kulattha*. Flesh of goat, domestic animals, water animals, buffalo from marshy regions and animals living in deserts. Liquor, old *Ghṛtam*, goat's milk and *Ghṛitam*, *Vāstuka*, leaves of of *Makoṭha*, brinjal, young radish, *Vyāghrī*, *Kāsaundi*, *Jīvantī* (leaves of *Toḍī*), leaves of *Śiriāṛi*, *Drākṣā*, *Bimbi*, *Jambīra* lemon, *Kamalakanda*, *Aḍuṣā*, smaller cardamom, cow urine, garlic, *Haraḍa*, *Tṛikaṭu* (dried ginger, *Pīppala*, *Marica*), lukewarm water, honey, poppy of paddy, sleeping during day time and easily digestible eatables. (B.R. 15/216-220)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures are harmful to the patient suffering from *Kāsa* : *Basti* therapy, nasal therapy, blood letting through leech or needle, exercising or wrestling, brushing teeth with twigs or powders, sun bathing, breathing bad odour, doing acts where dust gets into mouth and nose, excessive walking and walking in sand, various types of constipative eatables, taking *Vidāhī* and *Rūkṣa* types of meals, rice, holding the flow of vomit, *Kāsa* (coughs), belch and excretion, taking fish, *Kanda*, potato, leaves of mustard plant, *Tumbīphalā*, *Upodikā*, consuming old rice and water, excessive eating, eating heavy and cold rice and drinking water. (B.R. 15/221-224).

Differential Diagnosis of Doṣaja Kāsa

S. No.	***Vātaja***	***Pittaja***	***Kaphaja***	***Kṣataja***	***Kṣayaja***
Etiology	Intake of rough, cold & astringent food; consumption of food in less quantity; inadequate intake of food; fasting; excessive sexual intercourse; suppression of natural urges and excessive physical strain.	Excessive consumption of pungent, hot, *Vidahi*, sour & alkaline food, anger; exposure to the heat of fire & sun.	Consumption of heavy; *Abhiśyandi* (ingredients which cause obstruction to channels); sweet and unctuous substances; excessive sleep and sedentary lifestyle.	Excessive indulgence in sex; weight lifting; walking long distances; indulgence in fighting; combat with horses & elephants; studying in loud voice and injuries.	Consumption of irregular meals; unwholesome food; excessive indulgence in sex; suppression of natural urges; immensely hateful disposition and excessive worry.
Sputum & cough	Dry cough; patient feels difficulty in expectoration of sputum because of less quantity & dryness of sputum	Yellowishness of sputum & patient expectorates sputum mixed with *Pitta*	Chest is full of sputum and expectorates profuse, sweet, unctous & viscid sputum without pain.	Patient suffers from dry cough followed by expectoration of bloody sputum. While coughing patient produces cooing sound like that of a pigeon.	Patient expectorates sputum which is foul smelling, green or red in colour and appears like pus
Aggravation and reliving factors	Symptoms get relieved by foods & drinks which are unctous, sour, saline & warm. A symptom gets aggravated during empty stomach and diminution of strength, voice & *Ojas*.	*Pitta* aggravating factors	*Kapha* aggravating factors	Etiological factors mentioned under *Kṣataja kāsa*	Etiological factors mentioned under *Kṣayaja kāsa*
Prognosis	Curable	Curable	Curable	*Kṣataja Kāsa* is palliable if the patient is strong.	*Kṣayaja Kāsa* is curable in strong persons. If all the signs & symptoms present then it becomes difficult for treatment. Advanced stage of *Kṣayaja Kāsa* is incurable.
Modern correlation	Allergic bronchitis	Lower respiratory tract infections	Viral bronchitis, flu etc.	Acute bronchitis, traumatic lung injury, autoimmune lung diasease	Lung abscess, severe lower respiratory tract infections

MCQs

1. "*Durgandham, haritam, raktamsthivet and pooyopamamkapham*" are the characteristic feature of
   a. *Shosha* b. *Kshayaja Kasa*
   c. *Urahkshat* d. *Kshatajakasa*
2. According to *Charaka Samhita* select the types of *Kasaroga* indicating the severity in ascending order
   a. *Vataja, Pittaja, Shlesmaja, Kshayaja, Kshataja*
   b. *Vataja, Pittaja, Shlesmaja, Kshataja, Kshayaja*
   c. *Kshayaja, Kshataja, Shlesmaja, Vataja, Pittaja*
   d. *Kshayaja, Kshataja, Shlesmaja, Pittaja, Vataja*
3. *Paravataivakoojana* is the feature of
   a. *Kshatajakasa* b. *Kshayajakasa*
   c. *Vatikakasa* d. *Tamakaswasa*
4. स्थविराणां जराकासः indicates
   a. *Asadya* b. *Krichrasadya*
   c. *Yapya* d. *Anupakrama*
5. *Bhinnakamsyaswanatulyaghoshah* is observed in
   a. *Kshataksheena* b. *Kasa*
   c. *Swasa* d. *Apatantraka*
6. Sreemad darsana lochana is observed in
   a. *Pitta Kasa* b. *Kshataja Kasa*
   c. *Kshayaja Kasa* d. *Kapha Kasa*
7. Prognosis of *Kshataja kasa* in *Balavan*
   a. *Asadhya* b. *Sadhya*
   c. *Yapya* d. *Krichrasadya*
8. बहुलं मधुरं स्निग्धं निष्ठीवति घनं कफम् । कासमानो ह्यरुग् वक्षः सम्पूर्णमिव मन्यते । are the feature observed in
   a. *Rajayakshma* b. *Kshataja kasa*
   c. *Kshayaja Kasa* d. *Kaphaja kasa*
9. स पूर्वं कासते शुष्कं ततःष्ठीवेत् सशोणितम् । कण्ठेन रुजताऽत्यर्थं विरुग्णेनेव चोरसा् । are the features observed in
   a. *Raktha pitta* b. *Vatakasa*
   c. *Kshayaja Kasa* d. *Kshataja kasa*
10. दुर्गन्धं हरितं रक्तं ष्ठीवेत् पूयोपमं कफम् । are features observed in
    a. *Raktha pitta* b. *Vatakasa*
    c. *Kshayaja Kasa* d. *Kshataja kasa*
11. Prognosis of *Kshayaja kasa* in *Balavan*
    a. *Asadhya* b. *Sadhya*
    c. *Yapya* d. *Krichrasadya*

Answer

1.	b	2.	b	3.	a
4.	c	5.	b	6.	c
7.	c	8.	d	9.	d
10.	c	11.	c		

CHAPTER
15

Rajayakshma and Shosha

राजयक्ष्मा (Rājayakṣmā)

Introduction

Rājayakṣmā is caused by the simultaneous vitiation of all the three *doṣās as* a result of the following factors :

1. Suppression of the manifested natural urges
2. Reduction in the quantity of tissue elements as a result of waste of semen by excessive sex indulgence or because of fasting or poor nourishment.
3. Performance of activities much in excess of one's strength.
4. Irregularity in the intake of food or intake of unbalanced food.

The patient becomes emaciated because of the reduction in the quantity of tissue elements because of the obstruction to the channels of circulation of *rasa* (plasma or chyle) by all the three *doṣās* in aggravated condition dominated by *kapha*. Reduction of tissue elements also takes place as a consequence of excessive ejaculation of semen by sex indulgence.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

- The meaning of *Rājayakṣmā* is a group of signs and symptoms that occur together and characterize a particular abnormality or condition.
- The word *Rājayakṣmā* indicates a set of symptoms or conditions that occur together and suggest the presence of a certain disease or an increased chance of developing the disease.

यदा कष्टतमत्वात्तदा राजेव यक्ष्मा राजयक्ष्मेति ।
राज्ञो यक्ष्मा राजयक्ष्मा इति ॥
C.S.Ni. 6/12 (Cakarpāṇi)

राजा च यक्ष्मा च राजयक्ष्मा ततो मतः ।
यक्ष्माणां राजा राजयक्ष्मा इत्युक्तम् ॥
M.Ni. 10/1 (Madhu)

यक्ष्मा रोगो रोगाणां राजा अतो राजयक्ष्मेति ॥
S.S.Utt. 41/4-5 (Dalhaṇa)

The term *Rājayakṣmā* is comprises of two words *Rāja* and *Yakṣmā*. The word '*Rāja* ' means '*Diptau* ' which means the person who rules and the word '*Yakṣmā*' means '*Raga*' which means disease. Hence *Rājayakṣmā* means King among diseases *(Rājeva yakṣmā)* or King's (Moon-the King of *Nakṣatras* and *Dvijā)* disease which is most dreadful.

राज्ञश्चन्द्रस्य क्षयकार का यक्ष्मा राजा चासौ यक्ष्मा चेति वा ।
Śabdakalpdruma

यस्मात् स राज्ञः प्रागासीद्राजयक्ष्मा ततो मतः ॥
C.S.Ci. 8/11 (Cakarpāṇi)

राज्ञश्चन्द्रमसो यस्मादभूदेष किलामयः ।
तस्मात्तं राजयक्ष्मेति केचिदाहुः पुनर्जनाः ॥
Su.Utt. 41/5

यक्ष्माणां राजा राजयक्ष्मा यद्वा चन्द्रमासो जातत्वात् राजयक्ष्मेति ॥ *Sha.S. 7/22-23*

Yakṣmā first distressed the King *Candra* (Moon- King of planets), hence the learned scholars call it *Rājayakṣmā.*

यक्ष्म शब्देन च राजयक्ष्मवदने करोगयुक्तत्वं विकाराणां दर्शयति ॥ *Cakra.Ci.Ni. 1/5*

The word *Yakṣmā* indicates *syndrome*, like *Rājayakṣmā*

राजयक्ष्मा रोगसमूहानां *C.Sa. 25/40*

Rājayakṣmā is an aggregates or collection or group of diseases.

अनेकरोगानुगतो बहुरोगपुरोगमः ।
राजयक्ष्मा क्षयः शोषो रोगराडिति च स्मृतः ॥
A.H.Ni 5/1

राजयक्ष्मा क्षयः शोषो रोगराड़िति च स्मृतः ॥
S.K.D

Rajayakṣmā follows many diseases and many diseases arise from it. *Kṣaya, Śoṣa* and *Rogarāṭ* are the synonyms of the disease *Rājayakṣmā.*

रसः स्रोतः सु रुद्धेषु स्वस्थानस्थो विदह्यते ।
स ऊर्ध्वं कासवेगेन बहुरूपः प्रवर्तते ॥
जायन्ते व्याधयश्चातः षडेकादश वा पुनः ।
येषां सङ्घातयोगेन राजयक्ष्मेति कथ्यते ॥

C.Ci.8/43-44

Due to obstruction in the *Rasavāhi srotas,* the *Rasa dhātu* gets accumulated in its own place increases and then comes out with force of coughing from upper orifices in various forms. It is a recognizable complex of symptoms and physical findings which indicate a specific condition *Rājayakṣmā.*

Rājayakṣmā is a collection of recognizable clinical presentations of *Trirupa*, *Saḍrūpa* or *Ekādaśarūpa* or abnormalities that tend to occur together and are associated with a specific disease.

Mythological Origin of Rājayakṣmā

दिवौकसां कथयतामृषिभिर्वै श्रुता कथा ।
कामव्यसनसंयुक्ता पौराणी शशिनं प्रति ॥
रोहिण्यामतिसक्तस्य शरीरं नानुरक्षतः ।
आजगामाल्पतामिन्दोर्देहः स्नेहपरिक्षयात् ॥
दुहितॄणामसंभोगाच्छेषाणां च प्रजापतेः ।
क्रोधो निःश्वासरूपेण मूर्तिमान् निःसृतो मुखात् ॥
प्रजापतेर्हि दुहितॄरष्टाविंशतिमंशुमान् ।
भार्यार्थं प्रतिजग्राह न च सर्वास्ववर्तत ॥
गुरुणा तमवध्यातं भार्यास्वसमवर्तिनम् ।
रजःपरीतमबलं यक्ष्मा शशिनमाविशत् ॥
सोऽभिभूतोऽतिगुरुणा गुरुक्रोधेन निष्प्रभः ।
देवदेवर्षिसहितो जगाम शरणं गुरुम् ॥
अथ चन्द्रमसः शुद्धां मतिं बुद्ध्वा प्रजापतिः ।
प्रसादं कृतवान् सोमस्ततोऽश्विभ्यां चिकित्सितः ॥
स विमुक्तग्रहश्चन्द्रो विरराज विशेषतः ।
ओजसा वर्धितोऽश्विभ्यां शुद्धं सत्त्वमवाप च ॥
क्रोधो यक्ष्मा ज्वरो रोग एकार्थो दुःखसंज्ञकः ।
यस्मात् स राज्ञः प्रागासीद्राजयक्ष्मा ततो मतः ॥
स यक्ष्मा हुङ्कृवोऽश्विभ्यां मानुषं लोकमागतः ।
लब्ध्वा चतुर्विधं हेतुं समाविशति मानवान् ॥

C.S.Ci. 8/3-12

Mythological story in relation to the development of *Rājayakṣmā* was narrated by the gods to the sages i:e passionate indulgence of sex by *Candra* is said to be the prime factor for the manifestation of disease. King *Candra* exceedingly attached to *Rohiṇi* was reduced to loss of unctuousness and his carelessness towards health causes the development of *Rājayakṣmā.* Further, due to non-enjoyment with the rest of daughters of *Dakṣaprajāpati* became furious and his anger came out of his mouth in embodiment through expiration.

King *Candra* married 28 daughters of *Dakṣaprajāpati* but unable to satisfy all except *Rohiṇi.* Due to his discriminatory nature, filled up with *Rajas* and debility leading to genesis of *Rājayakṣmā.* Being subdued by the excessive anger of *Dakṣa,* the king *Candra* was depleted of his complexion. Lusterless *Candra* went to the refuge of the great accompanied by gods & godly sages. Being satisfied by about the good intentions of the moon, *Dakṣa* was pleased & as such he was treated by *Aśvins,* as a result king *Candra* vitality was enhanced & became free from disease and attained complexion and purity of mind.

Krodha, Yakṣmā, Jvara and *Roga* are all synonymous and denote *Dukha.* It afflicted first king *Candra,* hence termed as *Rājayakṣmā.* Later this disease afflicts human beings after being ousted by *Aśvins* having four etiological factors.

हेतु (Hetu)

इह खलु चत्वारि शोषस्यायतनानि भवन्ति; तद्यथासाहसं संधारणं क्षयो विषमाशनमिति । *C.S.Ni. 6/3*

क्षयाद्वेगप्रतीघातादाघाताद्विषमाशनात् । जायते कुपितैर्दोषैर्व्याप्तदेहस्य देहिनः ॥ *S.S.Utt. 41/9*

There a four etiological factors namely over exertion, suppression of natural urges, wasting and irregular dieting.

Sāhasa (over exertion) as etiological factor

तत्र साहसं शोषस्यायतनमिति यदुक्तं तदनुव्याख्यास्यामःयदा पुरुषो दुर्बलो हि सन् बलवता सह विगृह्णाति, अतिमहता वा धनुषा व्यायच्छति, जल्पति वाऽप्यतिमात्रम्, अतिमात्रं वा भारमुद्वहतिख अप्सु वा प्लवते चातिदूरम्, उत्सादनपदाघातने

वाऽतिप्रगाढमासेवते, अतिप्रकृष्टं वाऽध्वानं द्रुतमभिपतति, अभिहन्यते वा, अन्यद्वा किंचिदेवंविधं विषममतिमात्रं वा व्यायामजातमारभते, तस्यातिमात्रेण कर्मणोरः क्षण्यते। तस्योरः क्षतमुपप्लवते वायुः। स तत्रावस्थितः श्लेष्माणमुरःस्थमुपसंगृह्य पित्तं च दूषयन् विहरत्यूर्ध्वमधस्तिर्यक् च। तस्य योंऽशः शरीरसन्धीनाविशति तेनास्य जृम्भाऽङ्गमर्दो ज्वरश्चोपजायते, यस्त्वामाशयमभ्युपैति तेन रोगा भवन्ति उरस्या अरोचकश्च, यः कण्ठमभिप्रपद्यते कण्ठस्तेनोद्ध्वंस्यते स्वरश्चावसीदति, यः प्राणवहानि स्त्रोतांस्यन्वेति तेन श्वासः प्रतिश्यायश्च जायते, यः शिरस्यवतिष्ठते शिरस्तेनोपहन्यते; ततः क्षणनाच्चैवोरसो विषमगतित्वाच्च वायोः कण्ठस्य चोद्ध्वंसनात् कासः सततमस्य संजायते, स कासप्रसङ्गादुरसि क्षते शोणितं ष्ठीवति, शोणितागमनाच्चास्य दौर्बल्यमुपजायते; एवमेते साहसप्रभवाः साहसिकमुपद्रवाः स्पृशन्ति। ततः स उपशोषणैरेतैरुपद्रवैरुपद्रुतः शनैः शनैरुपशुष्यति। तस्मात् पुरुषो मतिमान् बलमात्मनः समीक्ष्य तदनुरूपाणि कर्माण्यारभेत कर्तुं; बलसमाधानं हि शरीरं, शरीरमूलश्च पुरुष इति॥

C.S.Ni. 6/4

When a weak person fights with a stronger person, or performs exercises with big bow or speaks too much or carries over weight, or swims for longer distances, or subjected to forceful anointing therapy or application of pressure by feet or runs fast to over long distance or subjects himself to assault or practices such other irregular regimens and physical exercise in excess as a result his chest becomes wounded. The wounded chest gets saturated with *Vāta*, and brings abnormality in both *Pitta* and *Kapha* and spreads upwards, down wards and sideways. After entering the joints causes yawning, malaise and fever. After entering the *Āmāśaya* produces diseases of chest and anorexia. After entering the throat produces irritation of the throat and hoarseness of voice. After entering *Prāṇavaha Srotas* causes dyspnoea and coryza. After entering the head produces distress in head. Thereafter, due to injury to chest patient constantly suffers from coughing due to irregular movement of *Vāta* and irritation of the throat. Frequent coughing leads to haemoptysis and weakness. These are the complications develops due to over excretion of an individual. Patients gradually develops cachexia day by day. That's why one has to perform various activities with due regard to his own strength because the body is dependent on strength & individual by the body.

Sandhāraṇa as etiological factor

संधारणं शोषस्यायतनमिति यदुक्तं तदनुव्या-ख्यास्यामःयदा पुरुषो राजसमीपे भर्तुः समीपे वा गुरोर्वा पादमूले द्यूतसभमन्यं वा सतां समाजं स्त्रीमध्यं वा समनुप्रविश्य यानैर्वाऽप्युच्चावचैरभियान् भयात् प्रसङ्गाद्धीमत्त्वाद्घृणित्वाद्वा निरुणद्ध्यागतान् वातमूत्रपुरीषवेगान् तदा तस्य संधारणाद्वायुः प्रकोपमापद्यते, स प्रकुपितः पित्तश्लेष्माणौ समुदीर्योर्ध्वमधस्तिर्यक् च विहरति; ततश्चांशविशेषेण पूर्ववच्छरीरावयवविशेषं प्रविश्य शूलमुपजनयति, भिनत्ति पुरीषमुच्छोषयति वा, पार्श्वे चातिरुजति, अंसाववमृद्नाति, कण्ठमुरश्चावधमति, शिरश्चोपहन्ति, कासं श्वासं ज्वरं स्वरभेदं प्रतिश्यायं चोपजनयति; ततः स उपशोषणैरेतैरुपद्रवैरुपद्रुतः शनैः शनैरुपशुष्यति। तस्मात् पुरुषो मतिमानात्मनः शारीरेष्वेव योगक्षेमकरेषु प्रयतेत विशेषेण; शरीरं ह्यस्य मूलं, शरीरमूलश्च पुरुषो भवति। *C.S.Ni. 6/6*

Due to suppression of impelled urges of flatus, urine and stool because of attending the king or the master or at the feet of the preceptor or while gambling or attending meetings of noble men or in the midst of women, or while travelling on uneven vehicle due to fear, environment, disgust leads to aggravation of *Vāta*. This excacerbated *Vāta* brings abnormality in *Pitta* and *Kapha* moves downwards, upwards & sideways. Vitiated *Vāta* spreads to different body parts and manifest symptoms like abdominal pain, diarrhoea or hardness of stools, excessive pain in the sides of the chest, shoulder; irritation in chest & throat, headache, cough, dyspnoea, fever, hoarseness of voice & coryza. These complications develops in *Sandhāraṇajanya Rājayakṣmā* patients and makes the person cachexic day by day. Wise person should adopt the strategy not to suppress the impelled urges in view to maintain good health.

Kṣaya as etiological factor (Anuloma kṣayaja hetu)

क्षयः शोषस्यायतनमिति यदुक्तं तदनुव्याख्यास्यामःयदा पुरुषोऽतिमात्रं शोकचिन्तापरिगतहृदयो भवति, ईर्ष्योत्कण्ठा-भयक्रोधादिभिर्वा समाविश्यते, कृशो वा सन् रूक्षान्नपानसेवी भवति, दुर्बलप्रकृतिरनाहारोऽल्पाहारो वा भवति, तदा तस्य

हृदयस्थायी रसः क्षयमुपैति; स तस्योपक्षयाच्छोषं प्राप्नोति, अप्रतीकाराच्चानुबध्यते यक्ष्मणा यथापदेक्ष्यमाणरूपेण ।

C.S.Ni. 6/7

- Excessive grief and worry afflicts heart
- Excessive envy, anxiety, fear, anger etc. leads to affliction in the heart.
- Excess consumption of rough food & drinks by emaciated persons.
- Less intake of food or excessive fasting by persons who are weak by nature.

All above etiological factors leads to diminution of *Rasa* which leads to manifestation of *Śoṣa* roga. Delay in management causes the genesis of *Rājayakṣmā.*

Kṣaya as etiological factor (Pratiloma kṣayaja hetu)

यदा वा पुरुषोऽतिहर्षादतिप्रसक्तभावः स्त्रीष्वति-प्रसङ्गमारभते, तस्यातिमात्रप्रसङ्गाद्रेतः क्षयमेति । क्षयमपि चोपगच्छति रेतसि यदि मनः स्त्रीभ्यो नैवास्य निवर्तते, तस्य चातिप्रणीतसङ्कल्पस्य मैथुनमाप-द्यमानस्य न शुक्रं प्रवर्ततेऽतिमात्रोपक्षीणरेतस्त्वात्, तथाऽस्य वायुर्व्यायच्छमानशरीरस्यैव धमनीरनुप्रविश्य शोणितवाहिनीस्ताभ्यः शोणितं प्रच्यावयति, तच्छुक्रक्षयादस्य पुनः शुक्रमार्गेण शोणितं प्रवर्तते वातानुसृतलिङ्गम् । अथास्य शुक्रक्षयाच्छोणित-प्रवर्तनाच्चसन्धयः शिथिलीभवन्ति, रौक्ष्यमुपजायते, भूयः शरीरं दौर्बल्यमाविशति, वायुः प्रकोपमापद्यते; स प्रकुपितो वशिकं शरीरमनुसर्पन्नुदीर्य श्लेष्मपित्ते परिशोषयति मांसशोणिते, प्रच्यावयति श्लेष्मपित्ते, संरुजति पार्श्वे, अवमृद्नात्यंसौ, कण्ठमुद्ध्वंसति, शिरः श्लेष्माणमुपत्क्लेश्य प्रतिपूरयति श्लेष्मणा, सन्धींश्च प्रपीडयन् करोत्यङ्गमर्दमरोचकाविपाकौ च, पित्तश्लेष्मोत्क्लेशात् प्रतिलोमगत्वाच्च वायुर्ज्वरं कासं श्वासं स्वरभेदं प्रतिश्यायं चोपजनयति; स कासप्रसङ्गादुरसि क्षते शोणितं ष्ठीवति, शोणितगमनाच्चास्य दौर्बल्यमुपजायते, ततः स उपशोषणैरेतैरुपद्रवैरुपद्रुतः शनैः शनैरुपशुष्यति । तस्मात् पुरुषो मतिमानात्मनः शरीरमनुरक्षञ्छुक्रमनुरक्षेत् । परा ह्येषा फलनिर्वृत्तिराहारस्येति ॥

C.S.Ni. 6/8

If person indulges excessive sexual intercourse due to excess passion & attachment leading to diminution of *Śukra*. If person doesn't care for his health at this level and further indulges in sexual intercourse, his semen will not be ejaculated because *Śukra* is already in diminished state, so due to excessive sexual act *Vāta* enters the blood vessels and causes blood discharge from seminal passage due to loss of *Śukra.* Aggravated *Vāta* manifests looseness in joints, roughness, weakness and exacerbation of *Vāta.* Vitiated *Vāta* spreading the entire body from where semen is exhausted and aggravating *Kapha* & *Pitta,* dries up the *Māmsa* and *Rakta,* eliminate *Kapha* & *Pitta,* causes pain in the sides of the chest and shoulders, irritation in throat, aggravate the *Kapha* in head; general malaise, anorexia, indigestion after afflicting the joints; due to vitiation of *Pitta* & *Kapha, Vāta* takes opposite course and manifest fever, cough, breathlessness, hoarseness of voice and coryza. Excess coughing leads to haemoptysis and injury in the chest. Patient becomes weak due to loss of blood & other complications leading to cachexia day by day. That's why wise men should preserve his semen to protect his body because it is the final outcome of the foods and drinks.

Viṣamāśana as etiological factor

विषमाशनं शोषस्यायतनमिति यदुक्तं, तदनुव्याख्यास्यामःयदा पुरुषः पानाशनभक्ष्यलेह्योपयोगान् प्रकृतिकरणसंयोगराशिदेशकालोपयोगसंस्थोपशयविषमानासेवते, तदा तस्य तेभ्यो वातपित्तश्लेष्माणो वैषम्यमापद्यन्ते; ते विषमाः शरीरमनुसृत्य यदा स्त्रोतसामयनमुखानि प्रतिवार्यावतिष्ठन्ते तदा जन्तुर्यद्यदाहारजातमाहरति तत्तदस्य मूत्रपुरीषमेवोपजायते भूयिष्ठं नान्यस्तथा शरीरधातुः; स पुरीषोपष्टम्भा-द्वर्तयति, तस्माच्छुष्यतो विशेषेण पुरीषमनुरक्ष्यं तथाऽन्येषामतिकृशदुर्बलानां; तस्यानाप्यायमानस्य विषमाशनोपचिता दोषाः पृथक् पृथगुपद्रवैर्युञ्जन्तो भूयः शरीरमुपशोषयन्ति; तत्र वातः शूलमङ्गमर्दं कण्ठोद्ध्वंसनं पार्श्वसंरुजनमंसावर्दं स्वरभेदं प्रतिश्यायं चोपजनयति; पित्तं ज्वरमतीसारमन्तर्दाहं च; श्लेष्मा तु प्रतिश्यायं शिरसो गुरुत्वमरोचकं कासं च, स कासप्रसङ्गादुरसि क्षते शोणितं निष्ठीवति, शोणितगमनाच्चास्य दौर्बल्यमुपजायते । एवमेते विषमाशनोपचितास्त्रयो दोषा राजयक्ष्माणमभिनिर्वर्तयन्ति । स तैरुपशोषणैरुपद्रवैरुपद्रुतः शनैः शनैः शुष्यति । तस्मात् पुरुषो मतिमान् प्रकृतिकरणसंयोगराशिदेशकालोपयोगसंस्थोपशयादविषममाहारमाहरेत् ॥ *C.S.Ni. 6/10*

If person adopts unhealthy dietetic pattern leads to manifestation of *Rājayakṣmā.* Consumption of drinks, eatables, chewables and lickable without paying proper attention in terms of nature, preparation, combination, quantity, locality, time, dietetic rules, suitability, his *Tridoṣa* get imbalanced. Imbalanced *Doṣās* spreads all over the body and when they get localized obstructing the opening of channels, then whatever food is consumed is mostly transformed into urine & faeces with less formation of *Dhātus*. Life of the person is sustained by retention of stool, that's why stool should be protected particularly in case of the individual suffering from consumption, excess emaciation and weakness. Accumulated *Doṣās* due to irregular dieting manifest various complications and lead to further depletion. *Vāta* causes various kinds of pain, general malaise, irritation in throat; pain in sides, grinding pain in shoulder, hoarseness of voice, rhinitis; *Pitta* produces fear, diarrhoea & burning sensation inside the body and *Kapha* produces coryza, heaviness in head, anorexia and cough. Excess coughing leads to chest injury & haemoptysis.

As a result persons become weak. Above complications lead to cachexia day by day due to drying up of all the *Dhātus*. Therefore, wise person should take food which is not unwholesome in terms of nature, preparation, combination, quantity, place, time, dietetic rules and wholesomeness.

राजयक्ष्मा निदान संप्राप्ति (Nidāna & Samprāpti of Rājayakṣmā)

कफप्रधानैर्दोषैर्हि रुद्धेषु रसवर्त्मसु ।
अतिव्यवायिनो वाऽपि क्षीणे रेतस्यनन्तरम् ॥
क्षीयन्ते धातवः सर्वे ततः शुष्यति मानवः ॥
S.S.Utt. 41/9-10

तैरुदीर्णोऽनिलः पित्तं कफं चोदीर्य सर्वतः ।
शरीरसन्धीनाविश्य तान् सिराश्च प्रपीडयन् ॥
मुखानि स्त्रोतसां रुद्ध्वा तथैवातिविवृत्य वा ।
सर्पन्नूर्ध्वमधस्तिर्यग्यथास्वं जनयेद् गदान् ॥
A.H.Ni. 5/5-6

Disease *Rājayakṣmā* manifests due to obstruction in *Rasavaha Srotas* due to *Kapha;* or in person who indulge too much in copulation as a result all the *Dhātus* gets depleted due to depletion of *Śukra* resulting in drying up of the body and manifest *Rājayakṣmā.*

Aggravated *Vāta* produces increase of both *Pitta* and *Kapha* spreads to all the joints of the body & to the *Sirās* ramify upwards, downwards, & sidewards, either obstructing or greatly dilating channels resulting in the manifestation of *Rājayakṣmā.*

रूपं त्वस्य यथोद्देशं निर्देक्ष्यामि सभेषजम् ॥
यथास्वेनोष्मणा पाकं शारीरा यान्ति धातवः ।
स्त्रोतसा च यथास्वेन धातुः पुष्यति धातुतः ॥
स्त्रोतसां संनिरोधाच्च रक्तादीनां च संक्षयात् ।
धातूष्मणां चापचयाद्राजयक्ष्मा प्रवर्तते ॥
तस्मिन् काले पचत्यग्निर्यदन्नं कोष्ठसंश्रितम् ।
मलीभवति तत् प्रायः कल्पते किञ्चिदोजसे ॥
तस्मात् पुरीषं संरक्ष्यं विशेषाद्राजयक्ष्मिणः ।
सर्वधातुक्षयार्तस्य बलं तस्य हि विड्बलम् ॥
रसः स्त्रोतःसु रुद्धेषु स्वस्थानस्थो विवर्धते ।
स उफर्ध्वं कासवेगेन बहुरूपः प्रवर्तते ॥
जायन्ते व्याधयश्चातः षडेकादश वा पुनः ।
येषां संघातयोगेन राजयक्ष्मेति कथ्यते ॥
C.S.Ci. 8/38-44

Dhātvāgnis metabolise the respective *Dhātus* and gets their nourishment by respective channels. But in case of *Rājayakṣmā* there is obstruction to their channels of circulation or if there is diminution of *Raktādi Dhātus* or depletion of *Dhātvāgnis* resulting in the manifestation of *Rājayakṣmā.* During this period whatever food is digested in the gastrointestinal tract by *Jaṭharāgni* is mostly converted to *Malās* and little amount contributes for the formation of *Ojas*. In *Rājayakṣmā* patients, more emphasis has been given to preservation of stool because in the state of depletion of *Dhātus,* the stool provides strength to the body. Due to blockade in the channels *Rasa* accumulates in its own place and becomes *Vidagdha. Rasadhātugata mala* comes out from upward channels in the form of cough etc. There after, six or eleven symptomatology or diseases are manifested and this congregation of symptomatology is known as *Rājayakṣmā.*

Anuloma Rājayakṣmā

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kaphādi doṣās*
Depletion of *Jaṭharāgni* causes formation of more *Malās* and little amount contributes for the formation of *Ojas*.

↓

Development of *Āma* inside the gastro intestinal tract

↓

Aggravated *Vāta* produces increase of both *Pitta* and *Kapha* spreads to all
the joints of the body & to the *Sirās* ramify upwards, downwards, &
sidewards, either obstructing or greatly dilating channels associated
with Obstruction in *Rasavaha Srotas*

↓

But in case of *Rājayakṣmā* there is obstruction to their channels of circulation or if there is diminution of *Raktādi Dhātus* or depletion of *Dhātvāgnis* resulting in the manifestation of *Rājayakṣmā*.

↓

Rājayakṣmā.

(In *Rājayakṣmā* patients, more emphasis has been given to preservation of stool because in the state of depletion of *Dhātus*, the stool provides strength to the body. Due to blockade in the channels *Rasa* accumulates in its own place and becomes *Vidagdha*. *Rasadhātugata mala* comes out from upward channels in the form of cough etc.)

Pratiloma Rājayakṣmā

Nidāna Sevana (Consumption of Etiological factors)

↓

Depletion of *Śukra Dhātu*

↓

Aggravation of *Vātādi doṣās*

↓

Aggravated *Vāta* produces increase of both *Pitta* and *Kapha* spreads to all the joints of the body & to the *Sirās* ramify upwards, downwards, & sidewards, either obstructing or greatly dilating channels

↓

Rājayakṣmā.

राजयक्ष्मा संप्राप्ति घटक (Saṁprāpti Ghaṭaka of Rājayakṣmā)

Doṣa : *Kapha Pradhāna Tridoṣaja*
Vāta → Vyāna Samāna, Apāna, Udāna & Prāṇa
Pitta → Pācaka, Sādhaka
Kapha → Kledaka, Bodhaka, Avalambaka

Dūṣya : *Dhātu → Rasa, Raktādi Dhātus*
Upadhātu → Sirā, Sandhi
Śārīrika mala → Mūtra, Puriṣa
Dhātumala → Kapha, Pitta, Sveda, Keśa, Loma, Nakha

Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*

Agniduṣṭi : *Mandatā*

Āma : *Āgnijanya*

Srotas : *Annavaha, Rasavaha & Śukravaha* mainly, *Rasavaha Srotas (Anuloma Kṣaya), Śukravaha Srotas (Pratiloma Kṣaya)* later all the srotas

Srotoduṣṭi : *Saṅga, & Vimārgagamana*
Udbhavasthāna : *Āmāśayottha & Pakvāśayottha*
Adhiṣṭhāna : *Sarvaśarīra*
Vyaktasthāna : *Mukha & Sarvaśarīra*
Sancārasthāna : *Rasavāhinyaḥ*
Svabhāva : *Cirakāri*
Prabhāva : Curable in strong
Incurable in emaciated & weak

Roga bheda :
1. *Pratiloma & Anuloma Rājayakṣmā*
2. Four types depending on etiological factors (*Sāhasaja, Sandhā-raṇaja, Viṣāmāṣanaja & Kṣayaja*

Roga Mārga : *Madhyama*

पूर्वरूप (Pūrvarupa)

तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा-प्रति-श्यायः, क्षवथुरभीक्ष्णं, श्लेष्मप्रसेकः, मुखमाधुर्यम्, अनन्नाभिलाषः, अन्नकाले चायासः, दोषदर्शनमदोषे-ष्वल्पदोषेषु वा भावेषु पात्रोदकान्नसूपापूपोपदंश-परिवेशकेषु, भुक्तवतश्चास्य हृल्लासः, तथोल्लेखन-मप्याहारस्यान्तरान्तरा, मुखस्य पादयोश्च शोफः, पाण्योश्चावेक्षणमत्यर्थम्, अक्ष्णोः श्वेतावभासता चातिमात्रं, बाह्वोश्च प्रमाणजिज्ञासा, स्त्रीकामता, निर्घृणित्वं, बीभत्सदर्शनता चास्य काये, स्वप्ने चाभीक्ष्णं दर्शनमनुदकानामुदकस्थानानां शून्यानां च ग्रामनगरनिगमजनपदानां शुष्कदग्धभग्नानां च वनानां कृकलासमयूरवानरशुकसर्पकाकोलूकादिभिः संस्पर्शनमधिरोहणं यानं वा श्वोष्ट्रखरवराहैः केशास्थिभस्मतुषाङ्गारराशीनां चाधिरोहणमिति (शोषपूर्वरूपाणि भवन्ति) ॥ *C.S.Ni. 6/13*

पूर्वरूपं प्रतिश्यायो दौर्बल्यं दोषदर्शनम् ।
अदोषेष्वपि भावेषु काये बीभत्सदर्शनम् ॥
घृणित्वमश्नतश्चापि बलमांसपरिक्षय ।
स्त्रीमद्यमांसप्रियता प्रियता चावगुण्ठने ॥
मक्षिकाघुणकेशानां तृणानां पतनानि च ।
प्रायोऽन्नपाने केशानां नखानां चाभिवर्धनम् ॥
पतत्रिभिः पतङ्गैश्च श्वापदैश्चाभिधर्षणम् ।
स्वप्ने केशास्थिराशीनां भस्मनश्चाधिरोहणम् ॥
जलाशयानां शैलानां वनानां ज्योतिषामपि ।
शुष्यतां क्षीयमाणानां पततां यच्च दर्शनम् ॥
प्राग्रूपं बहुरूपस्य तज्ज्ञेयं राजयक्ष्मणः ।
C.S.Ci. 8/33-38

The following prodromal signs and symptoms observed before the manifestation of Rājayakṣmā : *Rhinitis;* frequent sneezing; excess salivation, sweet taste in the mouth, aversion towards food; feeling of exhaustion during the meal time; findings fault in things which are free from fault or have little fault specially that of utensils, water, cereals, pulses, flour preparations, spicy preparations, things which are chewed before taking food *(Upadaṁśa)* and caterers, nausea after meals; intermittent vomiting during meal; swelling on the face & feet; frequent looking at the hands; excessive whitishness of eyes; curiosity about the measurement of arms, passionate attachment with women; cruel disposition; frightful appearance in his body.

The following things appear in dreams- seeing empty reservoirs; deserted villages, towns, cities and countries, dried, burnt and destroyed forest; coming in contact with chameleon, peacock, monkeys, parrots, serpents, crows, owls etc. or riding over dog, camels, donkeys, and pigs or vehicles drawn by them; climbing over heaps of hairs, bones, ash, chaff and charcoal; fall of planets; appearance of ugly signs & symptoms in the body; abhorence for eatables, diminution of strength & muscle tissue; attachments to alcohol, women & meat; desire for veiling; fall of flies, insects, hair & grass in food & drink; rapid growth of hair & nails; assault by birds, wasps & animals.

रूप (Rupa)

त्रिरूप राज्यक्ष्मा (Trirupa Rājayakṣmā)

त्रिभिर्वा पीडितं लिङ्गैर्ज्वरकासासृगामयैः ।
S.S.Utt. 41/15

The following are the three cardinal features of *Rājayakṣmā*

1. *Jvara* (Fever)
2. *Kāsa* (cough)
3. *Asrugamaya* (haemoptysis)

षड्रूप राजयक्ष्मा (Ṣaḍrupa Rājayakṣmā)

कासोंऽसतापो वैस्वर्यं ज्वरः पार्श्वशिरोरुजा ।
छर्दनं रक्तकफयोः श्वासो वर्चोगदोऽरुचिः ॥
रूपाण्येकादशैतानि यक्ष्मणः षडिमानि वा ।
कासो ज्वरः पार्श्वशूलं स्वरवर्चोगदोऽरुचिः ॥
सर्वैरर्धैस्त्रिभिर्वाऽपि लिङ्गैर्मांसबलक्षये ।
युक्तो वर्ज्यश्चिकित्स्यस्तु सर्वरूपोऽप्यतोऽन्यथा ॥
C.S.Ci. 8/45-47

Six cardinal signs & symptoms of *Rajayakṣmā* are as follows:

1. *Kāsa* (Cough)
2. *Jvara* (Fever)
3. *Pārśvasūla* (Pain in both sides)
4. *Svara gada* (Derangement of voice)
5. *Varcogada* (Diarrhoea)
6. *Arūci* (Anorexia/loss of appetite)

एकादश रूप (Ekādaśa Rupa)

Eleven cardinal signs & symptoms of *Rājayakṣmā* are as follows :

1. *Kāsa* (Cough)
2. *Ansatapa* (Burning sensation in shoulders)
3. *Vaiśvarya* (Derangement of voice)
4. *Jvara* (Fever)
5. *Parśvarūja* (Pain in both sides)
6. *Śirorūja* (Headache)
7. *Chardan Rakta* (Haemoptysis)
8. *Chardan Kapha* (Spitting of sputum)
9. *Śvasa* (Dyspnoea)
10. *Varcogada* (Diarrhoea)
11. *Arūci* (Anorexia/loss of appetite)

प्रतिश्याय (Pratiśyāya)-Coryza

घ्राणमूले स्थितः श्लेष्मा रूधिरं पित्तमेव वा।
मारुताध्मातशिरसो मारुतं श्यायते प्रति॥
प्रतिश्यायस्ततो घोरो जायते देहकर्शनः।
तस्य रूपं शिरःशूलं गौरवं घ्राणविप्लवः॥
ज्वरः कासः कफोत्क्लेशः स्वरभेदोऽरुचिः क्लमः।
इन्द्रियाणामसामर्थ्यं यक्ष्मा चातः प्रजायते॥
C.S.Ci. 8/48-50

Inflation of head with abnormal *Vāta, Kapha, Rakta* or *Pitta* situated at the root of nose rushes towards *Vāyu* as a result of which manifest severe *Pratiśyāya* and which causes emaciation of the body. Signs & symptoms of *Pratiśyāya* are headache, heaviness, stuffy nose, fever, cough, excessive sputum, hoarseness of voice, anorexia, fatigue and sense organs unable to perform their normal functions, lastly *Rājayakṣmā* manifests.

Nature of sputum

पिच्छिलं बहलं विस्त्रं हरितं श्वेतपीतकम्।
कासमानो रसं यक्ष्मी निष्ठीवति कफानुगम्॥
C.S.Ci. 8/51

Patient of *Rājayakṣmā* spits out sputum, which is slimy, thick, putrid in odour, green, white or yellow in colour.

Nature of fever

अंसपार्श्वाभितापश्च संतापः करपादयोः।
ज्वरः सर्वाङ्गगश्चेति लक्षणं राजयक्ष्मणः॥
C.S.Ci. 8/52

Characteristic feature of *Jvara* in *Rājayakṣmā*- i) Burning sensation in the shoulders & sides of the chest; ii) Burning sensation in the hands and feet and iii) Generalised fever. Some physicians consider these signs and symptoms as *trirupa rājayakṣmā.*

Nature of Voice

वातात्पित्तात्कफाद्रक्तात् कासवेगात् सपीनसात्।
स्वरभेदो भवेद्वाताद्रूक्षः क्षामश्चलः स्वरः॥
तालुकण्ठपरिप्लोषः पित्ताद्वक्तुमसूयते।
कफान्मन्दः विबद्धश्च स्वरः खुरखुरायते॥
सन्नो रक्तविबद्धत्वात् स्वरः कृच्छ्रात् प्रवर्तते।
कासातिवेगात् कषणः पीनसात्कफवातिकः॥
C.S.Ci. 8/53-55

Hoarseness of voice is caused abnormal *Vāta, Pitta, Kapha, Rakta,* impulse of coughing & chronic rhinits. If caused by *Vāyu,* the voice becomes rough, feeble & unstable; if affected by *Pitta* causes burning sensation in the palate & throat and aversion to speaking; if affected by *Kapha* the voice becomes slow, obstructed, and stertorous; if affected by *Rakta* the voice becomes low because of obstruction & voice comes out with difficulty. If it is affected by strain of excessive coughing as a result throat manifest abnormal voice. If affected by *Pinasa*, manifest mixed feature of *Kaphaja* & *Vātaja* features.

Nature of pain on sides

पार्श्वशूलं त्वनियतं संकोचायामलक्षणम्।
C.S.Ci. 8/56

Pain in the sides of chest is irregular sometimes, it is associated with constriction & expansion.

Nature of headache

शिरःशूलं ससंतापं यक्ष्मिणः स्यात्सगौरवम्॥
C.S.Ci. 8/56

Headache in *Rājayakṣmā* patients associated with burning sensation & heaviness.

Nature of blood

अभिसन्ने शरीरे तु यक्ष्मिणो विषमाशनात्।
कण्ठात्प्रवर्तते रक्तं श्लेष्मा चोत्क्लिष्टसंचितः॥
C.S.Ci. 8/57

Due to irregular meals patients of *Rājayakṣmā* becomes debilitated and agitated, & accumulated blood & sputum comes out from the throat.

Due to obstruction in *Raktavaha Srotas,* it does not reach *Māmsādi dhātus* & get accumulated in excess in *Āmāśaya* and comes out from throat.

Nature of breathlessness

वातश्लेष्मविबद्धत्वादुरसः श्वासमृच्छति ।
C.S.Ci. 8/59

Due to obstruction in *Prāṇavaha Srotas* by *Vāta* & *Kapha* patient of *Rājayakṣmā* suffers from dyspnoea.

Nature of diarrhoea

दोषैरूपहते चाग्नौ सपिच्छपतिसार्यते ॥
C.S.Ci. 8/59

Patient of *Rājayakṣmā* suffers from diarrhoea due to impaired *Agni* & vitiated *Doṣās* associated with slimyness.

Nature of Anorexia

पृथग्दोषैः समस्तैर्वा जिह्वाहृदयसंश्रितैः ।
जायतेऽरूचिराहारे द्विष्टैरर्थैश्च मानसैः ॥
कषायतिक्तमधुरैर्विद्यान्मुखरसैः क्रमात् ।
वाताद्यैररुचिं जातां मानसीं दोषदर्शनात् ॥
C.S.Ci. 8/60-61

Anorexia occurs due to involvement of single *Doṣā* or simultaneous vitiation of all the *Doṣās* in the region of tongue as well as heart. Psychic anorexia may manifest due to disgusting objects. Anorexia caused by *Vāta, Pitta* & *Kapha* should be identified by astringent, bitter and sweet tastes in mouth respectively. Psychic anorexia patients finds fault with substances.

Nature of Chardi

अरोचकात् कासवेगाद्दोषोत्क्लेशाद्भयादपि ।
छर्दिर्या सा विकाराणामन्येषामप्युपद्रवः ॥
C.S.Ci. 8/62

Chardi is caused by anorexia, impulse of coughing, excitement of *Doṣās* & fear and it may occur as complication in other diseases.

Doṣic predominance

स्वरभेदोऽनिलाच्छूलं संकोचश्चांसपार्श्वयोः ।
ज्वरो दाहोऽतिसारश्च पित्ताद्रक्तस्य चागमः ॥
शिरसः परिपूर्णत्वमभक्तच्छन्द एव च ।
कासः कण्ठस्य चोध्वंसो विज्ञेयः कफकोपतः ॥
S.S.Utt. 41/12-13

Hoarseness of voice, constriction of the shoulders and flanks and pain all over the body are due to aggravation of *Vāta,* excacerbated *Pitta* manifest fever, burning sensation, diarrhoea & haemoptysis, agitated *Kapha* produces fullness feeling in the head, lack of desire for food, cough and loss of function of the throat.

उपशय/पथ्य (Upaśaya/pathya)

Auṣadha : *Sitopalādi Cūrṇa, Durālabhādighṛta, Jivantyādyaghṛta, Tālisādya Cūrṇa & Guṭikā*

Anna : *Rājayakṣmā* patients should take corns and cereals which were harvested before one year, which are properly cooked, which are light, not devoid of potency, tasty and having good smell.

Foods & drinks which are nourishing, cooling, *Avidhāhi,* wholesome food, milk, ghee, meat etc.

Vihāra : Medicated bath by using *Jivaniya gaṇa dravyās,* apply perfume, wear garlands & ornaments, touch auspicious objects, offer prayer to the gods, physicians and Brahmins, massage, unction, external cleansing, and perform *Yajña* (sacrificial ceremony).

For a patient of consumption, the following diet and drinks are wholesome: 1. *Śāli* and *Ṣaṣṭhi* variety of rice, wheat, barley, *Mudga*, etc. 2. Different type of alcoholic drinks: and 3. Meat of animals, birds and different types of deer inhabiting *Jāṅgala* (arid) lands. If the patient is emaciated having diminished muscle tissue, then the nourishing meat of animals belonging to *Kravyādi* group (carnivorus animals) should be given. (B.R. 14/3-4)

It there is excessive aggravation of *Doṣās*, then the patient should be administered *Vamana* (emetic) and *Virecana* (purgation) therapies preceded by *Snehana* (oleation) and *Svedana* (fomentation) therapies. All these therapies should be administered with drugs which should have oleating and not a depleting effect. (B.R. 14/5)

In case of such a *Rājayakṣmā* patient that has become very lean and thin and who is attacked by several problems should be first treated with the *Vamana* (emetic), purgative, *Niruhaṇa*, *Anuvāsana* and *Svedana* therapies. The patient can be treated either with one or all of these therapies. However, it is not advised to treat a patient with *Pañcakarma* who has badly lost weight and strength. This might prove fatal to the patient. (B.R. 14/6)

Eating of the meats of animals and birds, the pulse of *Mudga* (kidney beans), red variety of rice, wheat, barley is helpful for patients of *Rājayakṣmā*. If the patient is well built, he should be administered with the *Virecana* (purgation) therapy. The patient should be served with the following edibles: wheat, *Mudga*, gram, mutton, milk of goat and its butter as well as *Ghṛta*, meat of *Kravyādya* (carnivorous) animals, meat of wild animals, dust of unbaked meats dried in sharp sun, *Kāmblika*, *Rāga Ṣāḍava* and *Veśavāra*. The moon light is helpful for the patient. He should stay in a place where goat's are tied. He should apply *Kalka* prepare out of urine and droppings of goat. He should also coat his body with sandalwood and other aromatic herbs. He should also bath with water treated with the aromatic herbs. The patient should wear good quality of clothes and jewelry. He should stay in palaces. He should sport garland of flowers. He should listen to as well as indulge in erotic discussions. He should also listen to instrumental music. He should stare at beautiful women. Helpful edibles for him also include banana fruits, jackfruit and mango, *Āmalakī*, date fruits, *Kamala Kanda*, *Paruṣka*, coconut, drum sticks, *Kāga Tindu*, palm fruits, raisin, aniseeds, rock salt, leaves of *Aḍuṣā*, *Ghṛta* prepared out of cow and buffalo milk, raw sugar and sweet dish called *Śikharīṇī*. The patient should offer sacrifices and donations to priests and Gods. Cardiac drinks and foods are also prescribed for the patients of *Rājayakṣmā*. (B.R. 14/318-325)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The eatables and avoidable in the case of Rājayakṣmā : Avoidable activities: suppression of natural urges hard labour, intercourse, sweating, waking in night hours, application of *Añjana*, eating tasteless meals, taking meals in odd hours, getting angry, performing hard exercises and the like. Avoidable eatables: eating melons, *Kulattha*, *Māṣa*, garlic bulbs, asafoetida, shoots of bamboo, *Urada* pulse, brinjal, bitter gourd, *Bilva* fruit, etc. (B.R. 14/326-329).

शोष (Śoṣa)

Introduction

Some other scholars say that *Soṣa* produces due to excessive sexual intercourse, grief, senility, physical activities, walking long distances, fasting, wounds & injury to the chest.

Causes of Śoṣa

व्यवायशोकस्थाविर्यव्यायामाध्वोपवासतः ।
व्रणोरःक्षतपीडाभ्यां शोषानन्ये वदन्ति हि ॥

S.S.Utt. 41/21

Some other scholars say that *Soṣa* produces due to excessive sexual intercourse, grief, senility, physical activities, walking long distances, fasting, wounds & injury to the chest.

व्यवाय शोषी (Vyavāya Śoṣī)

व्यवायशोषी शुक्रस्य क्षयलिङ्गैरुपद्रुतः ।
पाण्डुदेहो यथापूर्वं क्षीयन्ते चास्य धातवः ॥

S.S.Utt. 41/22

Śoṣa develops due to excessive sexual intercourse characterised by symptoms of *Śukrakṣaya* (exhaustion, *Daurbalya, Aśyaśoṣa, Aṅgamarda, Paṇḍutā* etc.), yellowish whitish discoloration of the body & person suffers from depletion of tissues in the preceding order.

शोक शोषी (Śoka Śoṣī)

प्रध्यानशीलः स्रस्ताङ्गः शोकशोष्यपि तादृशः ।
विना शुक्रक्षयकृतैर्विकारैरभिलक्षितः ॥

S.S.Utt. 41/23

Śoṣa develops due to excessive grief is characterised by the person who always looks like grief sticken, debility and features of *Śukrakṣaya* though not actually having *Śukrakṣaya*.

जरा शोषी (Jarā Śoṣī)

जराशोषी कृशो मन्दवीर्यबुद्धिबलेन्द्रियः।
कम्पनोऽरुचिमान् भिन्नकांस्यपात्रहतस्वरः॥
ष्ठीवति श्लेष्मणा हीनं गौरवारुचिपीडितः।
सम्प्रस्रुतास्यनासाऽक्षः शुष्करूक्षमलच्छविः॥
S.S.Utt. 41/24-25

Śoṣa develops due to old age is characterised by emaciation; loss of vitality, intelligence, strength; loss of normal functions of sense organs; tremors, loss of appetite, broken voice like the sound of bronze bell; spits devoid of sputum, heaviness of body, anorexia, more elimination of liquid from mouth, nose & eyes, numbness, rough & dirty complexion.

अध्व शोषी (Adhva Śoṣī)

अध्वप्रशोषी स्रस्ताङ्गः सम्भृष्टपरुषच्छविः।
प्रसुप्तगात्रावयवः शुष्कक्लोमगलाननः॥
S.S.Utt. 41/26

Śoṣa develops due to excessive walking is characterised by weakness of body, rough & lusterless complexion, absence of sensation in the body parts similar to a sleeping person, dryness of *Kloma*, throat and face.

व्यायाम शोषी (Vyāyāma Śoṣī)

व्यायामशोषी भूयिष्ठमेभिरेव समन्वितः।
उरःक्षतकृतैर्लिङ्गैः संयुक्तश्च क्षताद्विना॥
S.S.Utt. 41/27

Śoṣa develops due to excessive physical exercise is characterised by the same symptoms as described under *Adhva Śoṣī* in greater degree associated with symptoms of *Uraḥkṣata* (injury to chest) though not actually suffering from the same.

व्रण शोषी (Vraṇa Śoṣī)

रक्तक्षयाद्वेदनाभिस्तथैवाहारयन्त्रणात् ।
व्रणितस्य भवेच्छोषःस चासाध्यतमः स्मृतः॥
S.S.Utt. 41/28

Śoṣa develops in wounds due to excessive loss of blood, severe pain & restriction in the food intake & it is very difficult to treat.

उरक्षत शोषी (Urakṣata Śoṣī)

व्यायामभाराध्ययनैरभिघातिमैथुनैः ।
कर्मणा चाप्युरस्येन वक्षो यस्य विदारितम्॥
तस्योरसि क्षते रक्तं पूयः श्लेष्मा च गच्छति।
कासमानश्छर्दयेच्च पीतरक्तासितारुणम्॥
सन्तप्तवक्षाः सोऽत्यर्थं दूयनात्परिताम्यति।
दुर्गन्धवदनोच्छ्वासो भिन्नवर्णस्वरो नरः॥
S.S.Utt. 41/29-31

Urakṣata Śoṣa develops due to excessive physical exercises, lifting heavy weights, walking long distances, studying in loud pitch for longer period of time, injury, excessive sexual intercourse leads to injury in the chest. This leads to exudation of blood, pus, sputum via cough or vomiting, which are either yellow, red, white or mild red, associated with delusion, foul smell from the mouth & breath, loss of colour & voice.

MCQs

1. *Rogaraditi/rogarat* is synonym of
   a. *Rajayakshma* b. *Jwara*
   c. *Sannyasa* d. *Atatva-Abhinivesha*
2. *Shosha* is synonym of
   a. *Rajayakshma* b. *Jwara*
   c. *Sannyasa* d. *Atatva-Abhinivesha*
3. *Trirupa of* of *Rajayakshma* are
   a. *Jwara kasa aruchi*
   b. *Jwara swarabheda shirooruja*
   c. *Jwara shwasha atisara*
   d. *Jwara kasa asrugamaya*
4. There are trirupa, shadrupa & ekadsha rupa features observed in
   a. *Rajayakshma* b. *Jwara*
   c. *Sanyasa* d. *Atatvabhinivesha*
5. *Picchila, Bahala, Visram, Haritam, Swetapitakam* nature of sputum is observed in
   a. *Rajayakshama* b. *Kshayaja Kasa*
   c. *Kaphaja Kasa* d. *Tamak Shwasa*
6. *Akshnoh shwetavabhasata chatimatram* feature observed in purvarupa of
   a. *Pandu* b. *Rajayakshma*
   c. *Kasa* d. *Kamala*

7. *Keshanam nakhanam chabhivardanam* feature observed in purvarupa of
   - a. *Prameha* b. *Jwara*
   - c. *Rajayakshma* d. *Shwasha*

8. Shtree Madhya mamsa priyata feature observed in purvarupa of
   - a. *Prameha* b. *Jwara*
   - c. *Rajayakshma* d. *Shwasha*

9. कासो ज्वरः पार्श्वशूलं स्वरवर्चोगदोऽरुचितः are features observed in
   - a. *Parinam shoola* b. *Jwara*
   - c. *Rajayakshma* d. *Shwasha*

10. Which among the following is not the *Trirupa* of *Rajayakshma*
    - a. *Amsaparswaabhitapa*
    - b. *Karapadasantapa*
    - c. *Sarvangajwara*
    - d.*Swarabheda*

11. *Kshaya* is synonym of
    - a. *Rajayakshma* b. *Jwara*
    - c. *Sannyasa* d. *Atatva-Abhinivesha*

Answer

1.	a	2.	a	3.	d
4.	a	5.	a	6.	b
7.	c	8.	c	9.	c
10.	d	11.	a		

CHAPTER
16

Common Lung Disorder

Pneumonia

1. Pneumonia is defined as an acute respiratory illness associated with recently developed radiological pulmonary shadowing which may be segmental, lobar or multilobar.
2. Pneumonias are usually classified as community or hospital acquired, or those occurring in immune compromised hosts.
3. Lobar pneumonia is a radiological and pathological term referring to homogeneous consolidation of one or more lung lobes, often with associated pleural inflammation
4. Bronchopneumonia refers to more patchy alveolar consolidation associated with bronchial and bronchiolar inflammation often affecting both lower lobes.

Community-acquired pneumonia (CAP)

1. It is one of the lower respiratory tract infections.
2. The incidence varies with age, being much higher in the very young and very old, in whom the mortality rates are also much higher.
3. World-wide, CAP continues to kill more children than any other illness.
4. Most cases are spread by droplet infection and occur in previously healthy individuals but several factors may impair the effectiveness of local defences and predispose to CAP.
5. *Strep. Pneumonia* remains the most common infecting agent, and thereafter, the likelihood that other organisms may be involved depends on the age of the patient and the clinical context.
6. Viral infections are an important cause of CAP in children, and their contribution to adult CAP is increasingly recognised.

Factors that predispose to pneumonia

- Cigarette smoking
- Old age
- Upper respiratory tract
- Recent influenza infection

Infections

- Pre-existing lung disease
- Alcohol
- HIV
- Corticosteroid therapy
- Indoor air pollution

Clinical features

1. Fever
2. Rigor
3. Shivering
4. Vomiting predominate
5. The appetite is usually lost
6. Loss of appetite is usually present
7. Headache is common.
8. Pulmonary symptoms include
    - Breathlessness and cough, which at first is characteristically short, painful and dry, but later accompanied by the expectoration of mucopurulent sputum
    - Rust-coloured sputum may be seen in patients with *Strep. pneumoniae* infection,
    - Occasional patient may report haemoptysis.
    - Pleuritic chest pain may be a presenting feature and on occasion may be referred to the shoulder or anterior abdominal wall.

Physical examination findings

1. Upper abdominal tenderness is sometimes apparent in patients with lower lobe pneumonia or if there is associated hepatitis.
2. Proteinaceous fluid and inflammatory cells congest the airspaces, leading to consolidation of lung tissue
3. This improves the conductivity of sound to the chest wall and the clinician may hear

bronchial breathing and whispering pectoriloquy

4. Crackles are often also detected.

Investigations

X-ray :

1. A chest X-ray usually provides confirmation of the diagnosis.
2. In lobar pneumonia, a homogeneous opacity localised to the affected lobe or segment usually appears within 12-18 hours of the onset of the illness.
3. Radiological examination is helpful if a complication such as parapneumonic effusion, intrapulmonary abscess formation or empyema is suspected.

Microbiological tests

1. **Sputum:** direct smear by Gramand Ziehl-Neelsen stains.
2. **Culture** and antimicrobial sensitivity testing
3. **Blood culture:** frequently positive in pneumococcal pneumonia
4. **Serology:** acute and convalescent titres for *Mycoplasma, Chlamydia, Legionella,* and viral infections.
5. **Pneumococcal** antigen detection in serum or urine
6. **PCR:** *Mycoplasma* can be detected from swab of oropharynx Severe community-acquired pneumonia

   The above tests plus consider :
7. **Tracheal aspirate,** induced sputum, bronchoalveolar lavage, protected brush specimen or percutaneous needle aspiration. Direct fluorescent antibody stain for *Legionella* and viruses
8. **Serology:** *Legionella* antigen in urine. Pneumococcal antigen in sputum and blood. Immediate IgM for *Mycoplasma*
9. **Cold agglutinins:** positive in 50% of patients with *Mycoplasma*
10. A full range of microbiological tests should be performed on patients with severe CAP
11. The identification of *Legionella pneumophila* has important public health implications and requires notification.
12. In patients who do not respond to initial therapy, microbiological results may allow its appropriate modification.
13. Microbiology also provides useful epidemiological information.

Pulse oximetry

1. Pulse oximetry provides a non-invasive method of measuring arterial oxygen saturation (SaO2) and monitoring response to oxygen therapy SpO_2.

Arterial blood gas

1. An arterial blood gas is important in those with $SaO_2 < 93\%$ or with features of severe pneumonia, to identify ventilatory failure or acidosis.

Full blood count

1. The white cell count may be normal or only marginally raised in pneumonia caused by atypical organisms, whereas a neutrophil leucocytosis of more than 15×10^9/ L favours a bacterial aetiology.
2. A very high (> 20 x 109/1) or low (< 4 x 10y/ 1) white cell count may be seen in severe pneumonia.

Biochemical tests

1. Urea and electrolytes and liver function tests should also be checked.

Acute phase reactants

1. The C-reactive protein (CRP) is typically elevated.

Differential diagnosis of pneumonia

- Pulmonary infarction
- Pulmonary/pleural TB
- Pulmonary oedema (can be unilateral)
- Pulmonary eosinophilia
- Malignancy: bronchoalveolar cell carcinoma
- Rare disorders: cryptogenic organising

pneumonia/bronchiolitis obliterans organising pneumonia (COP/BOOP)

Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by airflow limitation that is not fully reversible. COPD includes emphysema, an anatomically defined condition characterized by destruction and enlargement of the lung alveoli; chronic bronchitis, a clinically defined condition with chronic cough and phlegm; and small airways disease, a condition in which small bronchioles are narrowed. COPD is present only if chronic airflow obstruction occurs; chronic bronchitis without chronic airflow obstruction is not included within COPD. It is the fourth leading cause of death and affects >10 million persons in the United States. COPD is also a disease of increasing public health importance around the world. Estimates suggest that COPD will rise from the sixth to the third most common cause of death worldwide by 2020.

Risk Factors

Cigarette Smoking; Airway Responsiveness; Respiratory Infections; Occupational Exposures; Ambient Air Pollution; Passive, or Second-Hand, Smoking Exposure; and Genetic Considerations.

Pathophysiology

Pathologic changes in chronic obstructive pulmonary disease (COPD) occur in the large (central) airways, the small (peripheral) bronchioles, and the lung parenchyma. Most cases of COPD are the result of exposure to noxious stimuli, most often cigarette smoke. The normal inflammatory response is amplified in persons prone to COPD development. The pathogenic mechanisms are not clear but are most likely diverse. Increased numbers of activated polymorphonuclear leukocytes and macrophages release elastases in a manner that cannot be counteracted effectively by antiproteases, resulting in lung destruction.

Clinical Features

Patients typically present with a combination of signs and symptoms of chronic bronchitis, emphysema, and reactive airway disease. Symptoms include the following: Cough, usually worse in the mornings and productive of a small amount of colorless sputum; Acute chest illness; Breathlessness: The most significant symptom, but usually does not occur until the sixth decade of life; Wheezing: May occur in some patients, particularly during exertion and exacerbations. The sensitivity of physical examination in detecting mild to moderate COPD is relatively poor, but physical signs are quite specific and sensitive for severe disease.

Physical Examination

In the early stages of COPD, patients usually have an entirely normal physical examination. Current smokers may have signs of active smoking, including an odor of smoke or nicotine staining of fingernails. Patients with severe airflow obstruction may also exhibit use of accessory muscles of respiration, sitting in the characteristic "tripod" position to facilitate the actions of the sternocleidomastoid, scalene, and intercostal muscles. Patients may develop cyanosis, visible in the lips and nail beds.

Advanced disease may be accompanied by systemic wasting, with significant weight loss, bitemporal wasting, and diffuse loss of subcutaneous adipose tissue. This syndrome has been associated with both inadequate oral intake and elevated levels of inflammatory cytokines Such wasting is an independent poor prognostic factor in COPD. Some patients with advanced disease have paradoxical inward movement of the rib cage with inspiration (Hoover's sign), the result of alteration of the vector of diaphragmatic contraction on the rib cage as a result of chronic hyperinflation.

Signs of overt right heart failure, termed *cor pulmonale*, are relatively infrequent since the advent of supplemental oxygen therapy.

Clubbing of the digits is not a sign of COPD, and its presence should alert the clinician to initiate an investigation for causes of clubbing. In this population, the development of lung cancer is the most likely explanation for newly developed clubbing.

Findings in severe disease include the following: Tachypnea and respiratory distress with simple activities; Use of accessory respiratory muscles and paradoxical indrawing of lower intercostal spaces (Hoover sign); Cyanosis; Elevated jugular venous pulse (JVP).

Peripheral edema and Thoracic examination reveals the following: Hyperinflation (barrel chest); Wheezing - Frequently heard on forced and unforced expiration; Diffusely decreased breath sounds; Hyperresonance on percussion; Prolonged expiration; Coarse crackles beginning with inspiration in some cases

Certain characteristics allow differentiation between disease that is predominantly chronic bronchitis and that which is predominantly emphysema.

Chronic bronchitis characteristics include the following: Patients may be obese; frequent cough and expectoration are typical; Use of accessory muscles of respiration is common; coarse rhonchi and wheezing may be heard on auscultation and Patients may have signs of right heart failure (ie, cor pulmonale), such as edema and cyanosis.

Emphysema characteristics include the following: Patients may be very thin with a barrel chest; Patients typically have little or no cough or expectoration; Breathing may be assisted by pursed lips and use of accessory respiratory muscles; patients may adopt the tripod sitting position; The chest may be hyperresonant, and wheezing may be heard; Heart sounds are very distant.

Ancillary Investigations

Spirometry : The formal diagnosis of COPD is made with spirometry; when the ratio of forced expiratory volume in 1 second over forced vital capacity (FEV1/FVC) is less than 70% of that predicted for a matched control; it is diagnostic for a significant obstructive defect.

Criteria for assessing the severity of airflow obstruction (based on the percent predicted post bronchodilator FEV1) are as follows:

- Stage I (mild): FEV1 80% or greater of predicted
- Stage II (moderate): FEV1 50-79% of predicted
- Stage III (severe): FEV1 30-49% of predicted
- Stage IV (very severe): FEV1 less than 30% of predicted or FEV1 less than 50% and chronic respiratory failure

Arterial blood gas (ABG) findings are as follows :

- ABGs provide the best clues as to acuteness and severity of disease exacerbation
- Patients with mild COPD have mild to moderate hypoxemia without hypercapnia
- As the disease progresses, hypoxemia worsens and hypercapnia may develop, with the latter commonly being observed as the FEV1 falls below 1 L/s or 30% of the predicted value
- pH usually is near normal; a pH below 7.3 generally indicates acute respiratory compromise
- Chronic respiratory acidosis leads to compensatory metabolic alkalosis

In patients with emphysema, frontal and lateral chest radiographs reveal the following:

- Flattening of the diaphragm; Increased retrosternal air space; A long, narrow heart shadow and Rapidly tapering vascular shadows accompanied by hyperlucency of the lungs
- Radiographs in patients with chronic bronchitis show increased bronchovascular markings and cardiomegaly

Advantages of high-resolution CT include the following:

- Greater sensitivity than standard chest radiography
- High specificity for diagnosing emphysema (outlined bullae are not always visible on a radiograph)
- May provide an adjunctive means of

diagnosing various forms of COPD (eg, lower lobe disease may suggest alpha1-antitrypsin (AAT) deficiency

Other tests are as follows:

- Hematocrit - Patients with polycythemia (hematocrit greater than 52% in men or 47% in women) should be evaluated for hypoxemia at rest, with exertion, or during sleep
- Serum potassium - Diuretics, beta-adrenergic agonists, and theophylline act to lower potassium levels
- Measure AAT in all patients younger than 40 years or in those with a family history of emphysema at an early age
- Sputum evaluation will show a transformation from mucoid in stable chronic bronchitis to purulent in acute exacerbations
- Pulse oximetry, combined with clinical observation, provides instant feedback on a patient's status
- Electrocardiography can help establish that hypoxia is not resulting in cardiac ischemia and that the underlying cause of respiratory difficulty is not cardiac in nature
- The distance walked in 6 minutes (6MWD) is a good predictor of all-cause and respiratory mortality in patients with moderate COPD; patients with COPD who desaturate during the 6MWD have a higher mortality rate than do those who do not desaturate
- Two-dimensional echocardiography can screen for pulmonary hypertension
- Right-sided heart catheterization can confirm pulmonary artery hypertension and gauge the response to vasodilators

Source - Harrisons Principles of Internal Medicine, 18th Edition and e medicine articles

Pleural effusion

The accumulation of serous fluid within the pleural space is termed pleural effusion.

Empyema

The accumulation of frank pus within the pleural space is termed empyema

Haemothorax

The accumulation of blood within the pleural space is termed haemothorax.

Chylothorax

The accumulation of chyle within the pleural space is termed chylothorax.

Causes of pleural effusion

Common causes :

- Pneumonia (para-pneumonic effusion)
- Cardiac failure
- Subdiaphragmatic disorders (subphrenic abscess, pancreatitis etc.)
- Tuberculosis
- Pulmonary infarction
- Malignant disease

Uncommon causes :

- Hypoproteinaemia (nephrotic syndrome, liver failure, malnutrition)
- Meigs syndrome (ovarian tumour plus pleural effusion)
- Connective tissue diseases (particularly systemic lupus erythematosus (SLE) and rheumatoid arthritis)
- Myxoedema
- Uraemia
- Asbestos-related benign pleural effusion
- Acute rheumatic fever
- Post-myocardial infarction syndrome

Pathogenesis

Pleural fluid accumulates

↓

As a result of either increased hydrostatic pressure or decreased osmotic pressure

↓

Or from increased microvascular pressure due to disease of the pleural surface itself or injury in the adjacent lung (exudative effusion)

↓

Pleural effusion

Clinical assessment

Pain on inspiration and coughing

Signs

1. Tachypnoea
2. Absent breath sounds and vocal resonance
3. Stony dullness on percussion
4. Crackles above effusion
5. Pleurisy (a pleural rub) often precedes the development of an effusion, especially in patients with underlying pneumonia, pulmonary infarction or connective tissue disease.
6. Breathlessness is the only symptom related to the effusion itself, and its severity depends on the size and rate of accumulation.

Investigations

Imaging :

1. The classical appearance of pleural fluid on the erect PA chest film is of a curved shadow at the lung base, blunting the costophrenic angle and ascending towards the axilla
2. Around 200 ml of fluid is required to be detectable on a PA chest X-ray
3. The smaller effusions can be identified by ultrasound or CT.
4. Previous scarring or adhesions in the pleural space can cause localised effusions.
5. Pleural fluid localised below the lower lobe ('subpulmonary effusion') simulates an elevated hemidiaphragm.
6. Fluid localised within an oblique fissure may produce a rounded opacity, simulating a tumour.

Ultrasonography :

7. It is more accurate than plain chest radiography for determining the volume of pleural fluid and frequently provides additional helpful information.
8. Visualisation of fluid facilitates skin marking to indicate a site for safe needle aspiration and guides pleural biopsy, increasing diagnostic yield.
9. The presence of loculation may suggest an evolving empyema or resolving haemothorax.
10. The technique may also distinguish pleural fluid from pleural thickening.

CT :

11. CT displays pleural abnormalities more readily than either plain radiography or ultrasound, and may distinguish benign from malignant pleural disease.

Pleural aspiration and biopsy :

12. In some clinical settings (e.g. left ventricular failure) it should not be necessary to sample fluid unless atypical features present.
13. Simple aspiration provides information on the colour and texture of fluid and on appearance alone may immediately suggest an empyema or chylothorax.
14. The presence of blood is consistent with pulmonary infarction or malignancy, but may represent a traumatic tap.

Biochemical analysis :

15. Biochemical analysis allows differentiation of transudates from exudates and Gram stain may indicted pneumonic effusion.

Cytological examination :

16. Cytological examination is essential, as the predominant cell type provides useful information.
17. A low pH suggests infection but may also be seen in rheumatoid arthritis, ruptured oesophagus or advanced malignancy.

Combining pleural aspiration with biopsy :

18. Combining pleural aspiration with biopsy increase the diagnostic yield, particularly when guided by either ultrasound or CT.

Thoracoscopy :

19. The best results are obtained from video-assisted thoracoscopy, allowing the operator to visualise the pleura and guide the biopsy directly

Light's criteria for distinguishing pleural transudate from exudate

Pleural fluid is an exudate if one more of the following criteria are met.

- Pleural fluid protein:serum protein ratio>0.5
- Pleural fluid LDH serum LDH ratio>0.6
- Pleural fluid LDH>two-thirds of the upper limit of normal serum LDH

(LDH=lactate dehydrogenase)

Bronchitis

Bronchitis is characterized by inflammation of the bronchial tubes (bronchi), the air passages that extend from the trachea into the small airways and alveoli.

Etiology

Respiratory viruses are the most common causes of acute bronchitis, and cigarette smoking is indisputably the predominant cause of chronic bronchitis.

Viral and bacterial infections in acute bronchitis

The most common viruses include influenza A and B, parainfluenza, respiratory syncytial virus, and coronavirus, although an etiologic agent is identified only in a minority of case.

Smoking and other causes of chronic bronchitis

Cigarette smoking is indisputably the predominant cause of chronic bronchitis. Common risk factors for acute exacerbations of chronic bronchitis are advanced age and low forced expiratory volume in one second (FEV1). Most (70-80%) acute exacerbations of chronic bronchitis are estimated to be due to respiratory infections.

Clinical Features

A complete history must be obtained, including information on exposure to toxic substances and smoking. Symptoms of bronchitis include the following: Cough (the most commonly observed symptom); Sputum production (clear, yellow, green, or even blood-tinged); Fever (relatively unusual; in conjunction with cough, suggestive of influenza or pneumonia); Nausea, vomiting, and diarrhoea (rare); General malaise and chest pain (in severe cases); Dyspnea and cyanosis (only seen with underlying chronic obstructive pulmonary disease [COPD] or another condition that impairs lung function); Sore throat; Runny or stuffy nose; Headache; Muscle aches and Extreme fatigue

Physical Examination Findings

Diffuse wheezes, high-pitched continuous sounds, and the use of accessory muscles (in severe cases). Diffuse diminution of air intake or inspiratory stridor (indicative of bronchial or tracheal obstruction). Sustained heave along the left sternal border (indicative of right ventricular hypertrophy secondary to chronic bronchitis). Clubbing on the digits and peripheral cyanosis (indicative of cystic fibrosis). Bullous myringitis (suggestive of mycoplasmal pneumonia). Conjunctivitis, adenopathy, and rhinorrhea (suggestive of adenoviral infection)

Ancillary Investigations

Bronchitis may be suspected in patients with an acute respiratory infection with cough; yet, because many more serious diseases of the lower respiratory tract cause cough, bronchitis must be considered a diagnosis of exclusion.

Complete blood count (CBC) with differential-

Procalcitonin levels (to distinguish bacterial from nonbacterial infections)

Sputum cytology (if the cough is persistent)

Blood culture (if bacterial super infection is suspected). Chest radiography (if the patient is elderly or physical findings suggest pneumonia). Bronchoscopy (to exclude foreign body aspiration, tuberculosis, tumors, and other chronic diseases)

Influenza tests
Spirometry
Laryngoscopy (to exclude epiglottitis)

Bronchiectasis

1. Bronchiectasis means abnormal dilatation of the bronchi.
2. Chronic suppurative airway infection with sputum production, progressive scarring and lung damage are present, whatever the cause.

Aetiology and pathogenesis

A.Congenital

1. Cystic fibrosis
2. Ciliary dysfunction syndromes

   Primary ciliary dyskinesia (immotile cilia syndrome)

   Kartagener's syndrome sinusitis and transposition of the Primary hypogammaglobulinaemia

B. Acquired: children

1. Pneumonia (complicating whooping cough or measles)
2. Primary TB
3. Inhaled foreign body

C. Acquired: adults

1. Suppurative pneumonia
2. Pulmonary TB
3. Allergic bronchopulmonary aspergillosis complicating asthma
4. Bronchial tumours

Pathogenesis

Bronchiectasis may result from a congenital defect affecting airway ion transport or ciliary function, such as cystic fibrosis

↓

Acquired secondary to damage to the airways by a destructive infection,
inhaled toxin or foreign body

↓

The result is chronic inflammation and infection in airways

↓

Chronic inflammatory and fibrotic changes are usually found in the surrounding lung tissue, resulting in progressive destruction of the normal lung architecture in advanced cases.

↓

Localised bronchiectasis may occur

↓

Due to the accumulation of pus beyond an obstructing bronchial lesion

↓

Such as enlarged tuberculous hilar lymph nodes, a bronchial tumour or
an inhaled foreign body (e.g. an aspirated peanut).

Clinical features

Symptoms of bronchiectasis :

1. Cough-Chronic productive cough due to accumulation of pus in dilated bronchi; usually worse in mornings and often brought on by changes of posture.
2. Sputum often copious and persistently purulent in advanced disease.
3. Halitosis is a common accompanying feature
4. Pneumonia and pleurisy occurs
5. Haemoptysis- Can be slight or massive and is often recurrent. Usually associated with purulent sputum or an increase in sputum purulence. Can, however, be the only symptom in so-called 'dry bronchiectasis
6. Poor general health-When disease is extensive and sputum persistently purulent, there may be associated weight loss, anorexia, lassitude, low-grade fever, and failure to thrive in children.
7. In these patients, digital clubbing is common

Physical signs

1. When there are large amounts of sputum in the bronchiectatic spaces, numerous coarse crackles may be heard over the affected areas.
2. Collapse with retained secretions blocking a proximal bronchus may lead to locally diminished breath sounds, with advanced disease may lead to scarring and overlying bronchial breathing.
3. Acute haemoptysis is an important complication of bronchiectasis

Investigations

A. *Bacteriological and mycological examination of sputum*

1. In addition to common respiratory pathogens, sputum culture may reveal *Pseudomonas aeruginosa,* fungi such as *Aspergiilus* and various mycobacteria.
2. Frequent cultures are necessary to ensure appropriate treatment of resistant organisms.

B. Radiological examination

1. Bronchiectasis, unless very gross, is not usually apparent on a chest X-ray.
2. In advanced disease, thickened airway walls, cystic bronchiectatic spaces, and associated areas of pneumonic consolidation or collapse may be visible.

C. CT

3. CT is much more sensitive, and shows thickened dilated airways (Fig. 19.28).

D. Assessment of ciliary function

Bronchial Asthma

The chronic airway inflammation, airflow obstruction and increased airway hyper-responsiveness leads to wheeze, cough, chest tightness and dyspnoea

Epidemiology

1. Current estimates suggest that asthma affects 300 million people world-wide and an additional 100 million persons will be diagnosed by 2025.
2. The socio-economic impact is enormous, particularly when poor control leads to days lost from school or work, unscheduled health-care visits and hospital admissions.

Etiology

1. Although the development, course of disease and response to treatment are influenced by genetic determinants
2. The rapid rise in the prevalence of asthma implies that environmental factors are critically important in terms of its expression.
3. The relationship between atopy (a propensity to produce IgE) and asthma is well established
4. Inhalation of an allergen into the airway include house dust mites, pets such as cats and dogs, pests such a cockroaches, and fungi (particularly Aspergillus) allergic bronchopulmonary aspergillosis,
5. Allergic mechanism are also implicated in some cases of occupational asthma
6. Although the aetiolgy of asthma is often elusive, an attempt should be made to identify the agents that may contribute to the appearance or aggravation of the condition.

7. With regard to potential allergens, particular enquiry should be made into exposure to a pet cat, guinea pig, rabbit or horse, pest infestation, or mould growth following water damage to a home or building

Pathophysiology

Chronic airway inflammation, airflow obstruction and increased airway hyper-responsiveness are the key components in the pathogenesis of asthma.

↓

Airway hyper-reactivity (AHR)-the tendency for airways to contract too easily and too much in response to triggers that have little or no effect in normal individuals

↓

Other factors likely to be important include the degree of airway narrowing and the influence of neurogenic mechanisms

↓

With increasing severity and chronicity of the disease, remodeling of the airway occurs

↓

Leading to fibrosis of the airway wall, fixed narrowing of the airway and a reduced response to bronchodilator medication

↓

Exercise-induced asthma

↓

Hyperventilation results in water loss from the pericellular lining fluid of the respiratory mucosa, which in turn triggers mediator release

↓

Heat loss from the respiratory mucosa may also

be important
↓
Persistent asthma
↓
A chronic and complex inflammatory response ensues
↓
It is characterized by an influx of numerous inflammatory cells
↓
The transformation and participation of airway structural cells
↓
The secretion of an array of cytokines, chemokines and growth factors
↓
Smooth muscle hypertrophy
↓
Hyperplasia
↓
Thickening of the basement membrane
↓
Mucous plugging
↓
Epithelial damage

Clinical features

1. Recurrent episodes of wheeze
2. Chest tightness
3. Breathlessness
4. Cough
5. Classical precipitants include exercise, particularly in cold weather, exposure to airborne allergens or pollutants, and viral upper respiratory tract infections.
6. Patients with mild intermittent asthma are usually asymptomatic between exacerbations.
7. Patients with persistent asthma report on-going breathlessness and wheeze, but variability is usually present with symptoms fluctuating over the course of one day, from day to day, or from month to month.
8. Asthma characteristically displays a diurnal pattern, with symptoms and lung function being worse in the early morning.
9. In some circumstances, the appearance of asthma is triggered by medications. For example, β-adrenceptor antagonists (β-blockers), ever when administered topically as eye drops, may induce bronchospasm, and aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) may also induce wheeze as above.

Diagnosis

1. The diagnosis of asthma is predominantly clinical and based on a characteristic history.
2. Supportive evidence is provided by the demonstration of variable airflow obstruction, preferably by using spirometry
3. The measurement of FEV1 and VC identify the obstructive nature of the ventilator defect, define its severity, and provide the basis of bronchodilator reversibility.
4. If spirometry is not available, a peak flow meter may be used.
5. Patients should be instructed to record peak flow readings after rising in the morning and before retiring in the evening.
6. A diurnal variation if PEF (the lowest values typically being recorded in the morning) of more than 20% is considered diagnostic and the magnitude of variability provides some indication of disease severity.
7. A trial of corticosteroids (e.g. 30 mg daily for 2 weeks) may be useful in documenting improvement in either FEV1 or PEF to establish the diagnosis.
8. When symptoms are predominantly related to exercise, an exercise challenge may be followed by a drop in lung function.
9. *Measurement of allergic status.* The presence of atopy may be demonstrated by skin prick tests.
10. The measurement of total and allergen-specific IgE.
11. A full blood picture may show peripheral blood eosinphilia.

12. *Radiological examination.* Chest X-ray appearances are often normal or show hyperinflation of lung fields. Lobar collapse may be seen if mucus occludes a large bronchus, and if accompanied by the presence of flitting infiltrates, may suggest that asthma has been complicated by allergic bronchopulmonary aspergillosis
13. An HRCT scan may be useful to detect bronchiectasis.

MCQs

1. Persistent reduction in forced expiratory flow rates associated with increases in the residual volume and the residual volume/total lung capacity ratio, nonuniform distribution of ventilation, and ventilation-perfusion mismatching is the most typical finding in
   a. COPD
   b. BA
   c. Lobar pneumonia
   d. Interstitial lung disease
2. Cough, weight loss, malaise, and gradual increase in dyspnea and inspiratory crackles and digital clubbing indicates
   a. Hypersensitivity Pneumonitis
   b. Tuberculosis
   c. Chronic obstructive pulmonary disease (COPD)
   d. Lung cancer
3. Patients with severe airflow obstruction may also exhibit use of accessory muscles ofrespiration, sitting in the characteristic tripod position to facilitate the actions of thesternocleidomastoid, scalene, and intercostal muscles. Patients may develop cyanosis,visible in the lips and nail beds indicates
   a. Bronchial Asthma
   b. Chronic Obstructive Pulmonary Disease
   c. Interstitial Lung Disease
   d. Pneumonia
4. Which among the following are obstructive lung disease
   I. Bronchial asthma
   II. Bronchiectasis
   III. Bronchiolitis
   IV. Pulmonary sarcoidosis
   a. I, II & III b. I, II & IV
   c. I, III&IV d. II, III& IV

Answer

1. a	2. a	3. b
4. a		

CHAPTER
17

Jvara Roga Nidana

Introduction of Jvara Roga Nidānam

Jvara born from anger of *Śambhu* produces severe harmful effects to the body like mitigation of strength, colour and digestive fire etc. It is considered as king of all diseases because it associates with many diseases. (*S.S.Utt. 39/323-324)*

Due to its pervading nature it affects all living creatures and difficult to tolerate, eventually leading to death of the patient.

Jvara manifests due to affliction of both *Śārīrika* and *Mānasika doṣās*. Living beings will not suffer from *Jvara* without involving these *Doṣās*. (C.S.Ci. 3/12)

Emaciation, feeling of darkness infront of eyes, sinful activities and death are the cardinal features of *Jvara* and it manifest due to his own actions leading to *Pañcatva* (death) (C.S.Ci. 3/13).

It originates from attachment *(Parigraha)* and also due to ferocious wrath of *Rudra* (C.S.Ci. 3/14)

Increased body temperature, anorexia, thirst, malaise, distress in cardiac region are the effects of *Jvara*. It is manifested in the form *Tamas* (entering into darkness) during the beginning of the birth and death. (C.S.Ci. 3/26-27).

It manifests due to wrath of *Maheśvara.* It kills all living beings. It produces great miseries for body, mind and sense organs. It greatly reduces intelligence, strength, complexion, joyfulness and enthusiasm. It induces exhaustion, exertion, unconsciousness and obstruction to food. It brings misery to the suffering individual, that's why it is called *Jvara*. It is a serious disease, having many complications and difficult for management. No other disease is powerful than *Jvara*. It is the reason behind giving nomenclature as king of all diseases. In different animals it is named differently. All living beings are born with *Jvara* and die with it. It induces delusiveness and the events of their previous life. Ultimately it takes away the life of the individual (C.S.Ci. 3/35)

ज्वरप्रत्यात्मिकं लिङ्गं सन्तापो देहमानसः ।
ज्वरेणाविशता भूतं न हि किञ्चिन्न तप्यते ॥
(C.S.Ci. 3/31)

Cardinal signs and symptoms of *Jvara* are increased body temperature associated with mental unpleasureness. It afflicts the body, mind and the sense organs.

No living being is spared by affliction of *Jvara*. That's why it is considered as most important disease and king of all diseases.

ज्वर स्वभाव (Jvara svabhāva)

शम्भुक्रोधोद्भवो घोरो बलवर्णाग्निसादकः ।
रोगराड् रोगसङ्घातो ज्वर इत्युपदिश्यते ॥
व्यापित्वात् सर्वसंस्पर्शात् कृच्छ्रत्वादन्तसम्भवात् ।
अन्तको ह्येष भूतानां ज्वर इत्युपदिश्यते ॥
(S.S. Utt. 39/323-324)

Fever manifest from the anger of Lord *Śambhu,* mitigator of strength, loss of complexion, loss of digestive fire, king of disease and group of many diseases. Because of its wide spreading, affecting all living beings, difficult to cure with the chance of causing death and it is the killer of all living beings.

Mythological theory in the development of *Jvara*

ज्वरमादौ प्रवक्ष्यामि स रोगानीकराट् स्मृतः ॥
रुद्रकोपाग्निसम्भूतः सर्वभूतप्रतापनः ।
तैस्तैर्नामभिरन्येषां सत्त्वानां परिकीर्त्त्यते ॥
जन्मादौ निधने चैव प्रायो विशति देहिनम् ।

अतः सर्वविकाराणामयं राजा प्रकीर्त्तितः ॥
ऋते देवमनुष्येभ्यो नान्यो विषहते तु तम् ।
कर्मणा लभते यस्माद् देवत्वं मानुषादपि ॥
पुनश्चैव च्युतः स्वर्गान्मानुष्यमनुवर्त्तते ।
तस्मात्ते देवभावेन सहन्ते मानुषा ज्वरम् ।
शेषाः सर्वे विपद्यन्ते तैर्य्यग्योना ज्वरार्दिताः ॥

(S.Utt. 39/8-13)

तमुवाचेश्वरः क्रोधं ज्वरो लोके भविष्यसि ।
जन्मादौ निधने च त्वमपचारान्तरेषु च ॥

(C.S.Ci. 3/25)

Jvara is the king of group of diseases. It is born from the wrathful fire of *Rudra (Vīrabhadra)*, torments all living beings is known by different names in the species of living creatures. For example, elephants etc., get suffered from this disease which is called *Kūṭa, Pākala* etc.

It afflicts the people in the beginning of birth and at the time of death. It also afflicts the person who adopts erratic life style and food regimen. That's why it is called king of all diseases.

ज्वर रोग हेतु (Hetu of Jvara Roga)

Etiological factors for the development of *Jvarās* are described are as follows:–

मिथ्याऽतियुक्तैरपि च स्नेहाद्यैः कर्मभिर्नृणाम् ।
विविधादभिघाताच्च रोगोत्थानात् प्रपाकतः ॥
श्रमात्क्षयादजीर्णाच्च विषात् सात्म्यर्त्तुपर्य्ययात् ।
ओषधीपुष्पगन्धाच्च शोकान्नक्षत्रपीडया ॥
अभिचाराभिशापाभ्यां मनोभूताभिशङ्कया ॥
स्त्रीणामपप्रजातानां प्रजातानां तथाऽहितैः ।
स्तन्यावतरणे चैवं ज्वरो दोषैः प्रवर्त्तते ॥

(S.S.Utt. 39/19-22)

Improper and excess administration of *Snehādi Samśodhana Karma*, various kinds of injury, suffering from diseases, suppuration, exertion, depletion of body tissues, improper digestion of food, poisons, changes of habitat and natural characteristics of the seasons, inhalation of smell of plants and flowers, sorrow, bad effects of stars and planets, effect of witchcraft and wrath of gods etc.; seizure *(Āveśa)* of an individual by evil spirits, abnormal delivery in women, and during production of breast milk in women. All above etiological factors brings disequilibrium state of *Doṣās.*

ज्वर रोग संप्राप्ति (Samprāpti of Jvara Roga)

दोषाः प्रकुपिताः स्वेषु कालेषु स्वैः प्रकोपणैः ।
व्याप्य देहमशेषेण ज्वरमापादयन्ति हि ॥
दुष्टाः स्वहेतुभिर्दोषाः प्राप्यामाशयमूष्मणा ।
सहिता रसमागत्य रसस्वेदप्रवाहिणाम् ॥
स्त्रोतसां मार्गमावृत्य मन्दीकृत्य हुताशनम् ।
निरस्य बहिरूष्माणं पक्तिस्थानाच्च केवलम् ॥
शरीरं समभिव्याप्य स्वकालेषु ज्वरागमम् ।
जनयन्त्यथ वृद्धिं वा स्ववर्णञ्च त्वगादिषु ॥

(S.S.Utt. 39/15-18)

Doṣās aggravates due to consumption of causative factors and during respective time spreads to entire body and produces *Jvara.* Aggravated *Doṣās* enters the *Āmāśaya* and combines with *Agni,* accompanying the *Rasa,* blocks the channels of *Rasa* and *Sveda,* impairs the functions of *Agni* and expels out the *Agni* from the site of digestion and spread to all over body and gives rise to *Jvara.* *Doṣās* exhibit its excacerbated symptoms and signs and brings abnormality in the skin, nail, eyes, tongue, urine and faeces.

ज्वर रोग संप्राप्ति घटक (Samprāpti ghaṭaka of Jvara Roga)

Doṣa : *Pitta Pradhāna Tridoṣaja.*
Vāta → Vyāna
Pitta → Pācaka
Kapha → Kledaka

Dūṣya : *Dhātu → Rasa*
Upadhātu → Tvacā
Śārīrikamala → Mūtra & Purīṣa
Dhātumala → Kapha, Sveda, Pitta

Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*

Agnidusṭi : *Mandāgni*

Āma : *Agnijanya*
Srotas : *Rasavaha, Svedavaha, Udakavaha*
Srotoduṣṭi : *Saṅga*
Adhiṣṭhāna : *Śarīra, Manas*
Sancārasthāna : *Rasavāhinyaḥ*
Udbhavasthāna : *Āmāśayottha*
Vyaktasthāna : *Tvacā*
Svabhāva : *Āśukāri* and *Cirakāri*
Prabhāva : *Ekadoṣaja,* recent onset-curable
Disease persist for long, involvement of all the three *Doṣās* having all the symptoms followed by loss of *Agni*- Incurable.
Roga Mārga : *Bāhya* and *Abhyantara*

पूर्वरूप (Pūrvarupa)

Premonitory symptoms and signs of *Jvara* are as follows:–

श्रमोऽरतिर्विवर्णत्वं वैरस्यं नयनप्लवः ।
इच्छाद्वेषौ मुहुश्चापि शीतवातातपादिषु ॥
जृम्भाऽङ्गमर्दो गुरुता रोमहर्षोऽरुचिस्तमः ।
अप्रहर्षश्च शीतञ्च भवत्युत्पत्स्यति ज्वरे ॥
सामान्यतो *(S.S.Utt. 39/25-27)*

तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथाममुखवैरस्यं, गुरुगात्रत्वम्, अनन्नाभिलाषः, चक्षुषोराकुलत्वम्, अश्र्वागमनं, निद्राधिक्यम्, अरतिः, जृम्भा, विनामः, वेपथुः, श्रमभ्रमप्रलापजागरणरोमहर्षदन्तहर्षाः, शब्दशीतवातातपसहत्वासहत्वम्, अरोचकाविपाकौ, दौर्बल्यम्, अङ्गमर्दः, सदनम्, अल्पप्राणता, दीर्घसूत्रता, आलस्यम्, उचितस्य कर्मणो हानिः, प्रतीपता स्वकार्येषु, गुरूणां वाक्येष्वभ्यसूया, बालेभ्यः प्रद्वेषः, स्वधर्मेष्वचिन्ता, माल्यानुलेपनभोजन- परिक्लेशनं, मधुरेभ्यश्च भक्षेभ्यः प्रद्वेषः, अम्ललवणकटुक- प्रियता च, इति ज्वरस्य पूर्वरूपाणि भवन्ति प्राक्संतापात्; अपि चैनं संतापार्तमनुबध्नन्ति॥ *(C.S.Ni. 1/33)*

Fatigue, restlessness, discoloration, loss of taste in mouth, lachrymation, liking and disliking of sound, cold, wind and sun alternatively often; yawning, malaise, heaviness, horripilations, loss of taste, laziness, absence of pleasure, feeling of cold, loss of appetite, congestion in the eyes, excessive sleep, dislike for work, bending, tremors, giddiness, delirium, awakening in the night, sensation in teeth, indigestion, lassitude, debility, diminished vitality, dilatory tendency, laziness, sluggishness in routine activities, aversion towards own activities, disregard to the instructions of elders, disliking for children, unmindful towards own duties, lack of interest for the use of garland, ointments and food; aversion towards sweet food, liking for sour, saline and pungent things.

Above symptoms continue to exist after the complete manifestations of *Jvara.*

विशेष पूर्वरूप (Viśeṣa pūrvarupa)

विशेषात्तु जृम्भाऽत्यर्थं समीरणात् ।
पित्तान्नयनयोर्दाहः, कफान्नान्नभिनन्दनम् ॥
सर्वलिङ्गसमवायः सर्वदोषप्रकोपजे ।
द्वयोर्द्वयोस्तु रूपेण संसृष्टं द्वन्द्वजं विदुः ॥
(S.S.Utt. 39/27-28)

If yawning is predominant feature, then it indicates forthcoming disease is *Vātaja Jvara.* If burning sensation is predominant clinical feature then it indicates prodromal symptoms of *Pittaja Jvara.*

Dislike for food is predominant symptom indicates premonitory symptom of *Kaphaja Jvara.* Mixed features are observed in combination of two and three *Doṣās* involvement.

ज्वरभेद (Jvarabheda)

दौषैः पृथक् समस्तैश्च द्वन्द्वैरागन्तुरेव च ।
अनेककारणोत्पन्नः स्मृतस्त्वष्टविधो ज्वरः ॥
(S.S.Utt. 39/14-15)

द्विविधो विधिभेदेन ज्वरः शारीरमानसः ।
पुनश्च द्विविधो दृष्टः सौम्यश्चाग्नेय एव वा ॥
अन्तर्वेगो बहिर्वेगो द्विविधः पुनरुच्यते ।
प्राकृतो वैकृतश्वैव साध्यश्चासाध्य एव च ॥
पुनः पञ्चविधो दृष्टो दोषकालबलाबलात् ।
संततः सततोऽन्येद्युस्तृतीयकचतुर्थकौ ॥
पुनराश्रयभेदेन धातूनां सप्तधा मतः ।
भिन्नः कारणभेदेन पुनरष्टविधो ज्वरः ॥
(C.S.Ci. 3/32-35)

Jvara is classified into different categories are as follows:

1. *Śārīraja* (Physical) and *Mānasika* (Mental)
2. *Saumya* (caused by Cold) and *Āgneya* (caused by Heat)
3. *Antarvega* (Internal) and *Bahirvega* (External)
4. *Prākṛta* (Natural) and *Vaikṛta* (Unnatural)
5. *Sādhya* (Curable) and *Asādhya* (Incurable)

It is classified into five types depending on strength and weakness of *Doṣās and time.*

(a) *Santata*
(b) *Satata*
(c) *Anyeduṣka*
(d) *Tṛtīyaka*
(e) *Caturthaka*

According to location in *Dhātus,* it is classified into varieties of *Dhātugata Jvara.*

(a) *Rasadhātugata Jvara*
(b) *Rakta dhātugata Jvara*
(c) *Māmsa dhātugata Jvara*
(d) *Medo dhātugata Jvara*
(e) *Asthidhātugata Jvara*
(f) *Majjādhātugata Jvara*
(g) *Śukradhātugata Jvara*

Depending on causes it is classified into eight types.

(a) *Vātaja Jvara*
(b) *Pittaja Jvara*
(c) *Kaphaja Jvara*
(d) *Vāta-Pittaja Jvara*
(e) *Vāta-Kaphaja Jvara*
(f) *Pitta-Kaphaja Jvara*
(g) *Vāta-Pitta-Kaphaja Jvara*
(i) *Āgantuja Jvara*

शारीरज एवं मानसिक ज्वर (Śārīraja evam Mānasika Jvara)

शारीरो जायते पूर्वं देहे, मनसि मानसः ।
वैचित्त्यमरतिर्ग्लानिर्मनसस्तापलक्षणम् ॥
इन्द्रियाणां च वैकृन्यं ज्ञेयं(देह)संतापलक्षणम् ।

(C.S.Ci. 3/36-37)

Śārīraja Jvara manifests in the body due to affliction of *Śārīrika Doṣās. Mānasika Jvara* arises in the body due to affliction of *Mānasika Doṣās. Tāpa* to the mind produces mental distraction, restlessness and feeling of weakness. *Santāpa* to the sense organs manifest abnormality in the sense organs.

सौम्य एवं आग्नेय ज्वर (Saumya evam Āgneya Jvara)

वातपित्तात्मकः शीतमुष्णं वातकफात्मकः ॥
इच्छत्युभयमेतत्तु ज्वरो व्यामिश्रलक्षणः ।
योगवाहः परं वायुः संयोगादुभयार्थकृत् ॥
दाहकृत्तेजसा युक्तः, शीतकृत् सोमसंश्रयात् ।

(C.S.Ci. 3/37-39)

Patients of *Vātaja-Pittaja Jvara* crave for cold things; patients of *Vātaja-Kaphaja Jvara* desires for hot things; During amalgamation of *doṣās* mixed symptoms are manifested.

Due to synergistic actions of *Vāta,* it produces both types of effects, for example in combination with *Pitta* produces heat and in conjunction with *Kapha* produces cold.

अन्तर्वेग एवं बहिर्वेग ज्वर (Antarvega evam Bahirvega Jvara)

अन्तर्दाहोऽधिकस्तृष्णा प्रलापः श्वसनं भ्रमः ॥
सन्ध्यस्थिशूलमस्वेदो दोषवर्चोविनिग्रहः ।
अन्तर्वेगस्य लिङ्गानि ज्वरस्यैतानि लक्षयेत् ॥
संतापोऽभ्यधिको बाह्यस्तृष्णादीनां च मार्दवम् ।
बहिर्वेगस्य लिङ्गानि सुखसाध्यत्वमेव च ॥

(C.S.Ci. 3/39-41)

Antarvega Jvara is characterised by burning sensation inside the body, thirst, delirium, difficulty in breathing, giddiness, pain in joints and bones, absence of perspiration, non elimination of *Doṣās* and stool.

Bahirvegaja Jvara is characterised by excessive temperature in the exterior parts of the body and mildness of thirst etc. and it is curable.

प्राकृत ज्वर एवं वैकृत ज्वर (Prākṛta Jvara evam Vaikṛta Jvara)

कालप्रकृतिमुद्दिश्य निर्दिष्टः प्राकृतो ज्वरः ॥
प्रायेणानिलजो दुःखः कालेष्वन्येषु वैकृतः ।
हेतवो विविधास्तस्य निदाने संप्रदर्शिताः ॥

(C.S.Ci. 3/48-49)

प्राकृत ज्वर (Prākṛta Jvara)

The *Jvara* manifested in the *Vasantha* and *Śarad ṛtu* are called *Prākṛta Jvarās* and these are easily curable. During *Śarad ṛtu pitta* aggravates because of environment which supports its aggravation like hotness etc. Accumulated *Kapha* in the cold season gets aggravated in the *Vasanta.*

Accumulation of *Pitta* during rainy season due to sourness in *Vipāka* of water and drugs. *Pitta* gets excited during *Śarad* due to intense heat of sun and manifest *Pittaja Jvara* and *Kapha Doṣa* is the secondary associate. *Śarad ṛtu* included under *Visarga Kāla* i:e the period in which person becomes physically strong and patients may sustain the fasting without creating any problem as a result patients cures from disease.

Kapha gets accumulated during winter *(Hemanta)* because of the sweet *Vipāka* of water and drugs. It becomes excited during spring season *(Vasanta)* due to effect of strong rays of *Sun* leading to manifestation of *Kaphaja Jvara.* In this case, *Vāta* and *Kapha* are the secondary associates. It is manifested during *Ādāna Kāla.*

Management of *Prākṛta Jvara* i:e *Pittaja* and *Kaphaja Jvara* can be done successfully after considering the strength and weakness of the *Doṣās* in the beginning, middle and end of the seasons. Depending upon the seasonal effects *Prākṛta Jvara* is manifested and it is curable except *Vātaja Jvara. Prākṛta Vātaja Jvara* is considered as difficult for management due to mutual contradiction in the line of treatment of *Jvara.*

वैकृत ज्वर (Vaikṛta Jvara)

Jvara manifest in seasons other than that responsible for vitiation of the concerned *Doṣa* is understood as *Vaikṛta Jvara.* For example *Kaphaja Jvara* during *Śarad ṛtu, Pittaja Jvara* during *Vasanta ṛtu* etc. and it is very difficult for management.

साध्य एवं असाध्य ज्वर (Sādhya evam Asādhya Jvara)

वलवत्स्वल्पदोषेषु ज्वरः साध्योऽनुपद्रवः।
हेतुभिर्वहुभिर्जातो वलिभिर्वहुलक्षणः॥
ज्वरः प्राणान्तकृद्यश्च शीघ्रमिन्द्रियनाशनः।
सप्ताहाद्वा दशाहाद्वा द्वादशाहात्तथैव च॥
सप्रलापभ्रमश्वासस्तीक्ष्णो हन्याज्ज्वरो नरम्।
ज्वरः क्षीणस्य शूनस्य गम्भीरो दैर्घरात्रिकः॥
असाध्यो वलवान् यश्च केशसीमन्तकृज्ज्वरः।
(C.S.Ci. 3/50-53)

साध्य ज्वर (Sādhya Jvara)

Strong physical strength, little morbidity of *Doṣās* and free from complications is the characteristic feature indicates curable nature of *Jvara.*

असाध्य ज्वर (Asādhya Jvara)

The following factors indicates the incurability or bad prognosis of *Jvara* patients:–

- Manifestation of *Jvara* due to many etiological factors
- Complete appearance of signs and symptoms
- Quick destruction of sense organs.
- Jvara associated with delirium, giddiness, breathlessness kills the patient either on 7th day or 10th day or 12th day.
- Manifestation of *Jvara* in weak and edematous persons.
- *Jvara* which is deep seated (spreading to all *Dhātus*)
- Persistence of *Jvara* for several nights.
- Hairs of the head fall apart to produce a straight line.

विषमज्वर (Viṣamajvara)

विषमो विषमारम्भक्रियाकालोऽनुषङ्गवान्।
(A.H.Ni. 2/69)

Viṣamajvara is characterised by irregular onset, inconsistent symptoms and appear in irregular time; and it persists for longer duration.

विषमज्वर हेतु (Viṣamajvara Hetu)

Causative factors for the production of ***Viṣamajvara*** **are as follows:–**

कृशानां व्याधिमुक्तानां मिथ्याहारादिसेविनाम्।
अल्पोऽपि दोषो दूष्यादेर्लब्ध्वाऽन्यतमतो वलम्॥
सविपक्षो ज्वरं कुर्याद्विषमं क्षयवृद्धिभाक्।

दोषः प्रवर्तते तेषां स्वे काले ज्वरयन् बली ॥
निवर्तते पुनश्चैव प्रत्यनीकबलाबलः ।

(*A.H.Ni. 2/64-65*)

केचिद् भूताभिषङ्गोत्थं ब्रुवते विषमज्वरम् ॥

(*S.S.Utt. 39/68*)

Emaciated persons, one who recently relieved from disease indulging in unsuitable dietetics and life style etc., even the mildly vitiated *Doṣās,* gain strength from the *Dūṣyās (Dhātu, Mala, Kāla)* and manifest *Viṣamajvara.* Some people say that it manifest due to invasion of *Bhūtās* (evil spirits) into the body.

Here symptoms exhibited are either increased or decreased in a particular period of time. Symptoms appear when the *Doṣās* are strong during their own respective time and disappears at the time when the *Doṣās* are made weak by the strength of the other factors like *dhātus, malās,* habitat, season etc.

क्षीणे दोषे ज्वरः सूक्ष्मो रसादिष्वेव लीयते ॥
लीनत्वात्कार्श्यवैवर्ण्यजाड्यादीनादधाति सः ।

(*A.H.Ni. 2/66-67*)

Doṣās which manifest *Viṣamajvara* become decreased, the *Jvara* becomes mild and lurks in the *Rasādi Dhātus* leading to emaciation, discolouration, lassitude etc.

आसन्नविवृतास्यत्वात्स्रोतसां रसवाहिनाम् ॥
आशु सर्वस्य वपुषो व्याप्तिर्दोषेण जायते ।
सन्ततः सततस्तेन विपरीतो विपर्ययात् ॥

(*A.H.Ni. 2/67-68*)

Being superficial and openness or clearance of the path of *Rasavaha srotas* leads to spreading of *Doṣās* throughout the body as a result continuous variety of *Santata Jvara* manifest without remissions. On the contrary remaining *Satata, Anyedhyuṣka, Tṛtīyaka, Caturthaka* and *Caturthaka Viparyaya* characterised by intermittent *Jvara* having remissions at a particular period of time.

रात्र्यह्नोः षट्सु कालेषु कीर्त्तितेषु यथा पुरा ।
प्रसह्य विषमोऽभ्येति मानवं बहुधा ज्वरः ॥
स चापि विषमो देहं न कदाचिद् विमुञ्चति ।
ग्लानि-गौरव-कार्श्येभ्यः स यस्मान्न प्रमुच्यते ।
वेगे तु समतिक्रान्ते गतोऽयमिति लक्ष्यते ॥
धात्वन्तरस्थो लीनत्वान्न सौक्ष्म्यादुपलभ्यते ।
अल्पदोषेन्धनः क्षीणः क्षीणेन्धन इवानलः ॥

(*S.S.Utt. 39/62-65*)

Viṣamajvara manifest at the six periods of day and night i:e fore noon, midday, afternoon, evening, midnight and early morning by expressing powerful symptoms and troubles to the patients in many ways. It do not leave the patients completely and remains for longer period of time. It produces exhaustion, heaviness, emaciation; patient feels relieved after the pacification of bouts, but *Jvara* remains lurks inside the body i:e inside the *Dhātus* in a minute form like a spark of fire with little fuel.

दोषोऽल्पोऽहितसम्भूतो ज्वरोत्सृष्टस्य वा पुनः ।
धातुमन्यतमं प्राप्य करोति विषमज्वरम् ॥

(*S.S.Utt. 39/66*)

Mild aggravation of *Doṣās* by incompatible dietetics and behaviour produces *Viṣamajvara* after invading any one of the *Dhātus.*

Development of *Viṣamajvara* by involving *Dhātu*

Residing in *Rasadhātu* manifest *Santata Jvara*

Residing in *Raktadhātu* manifest *Satatajvara*

Residing in *Māmsadhātu* manifest *Tṛtīayaka Jvara*

Residing in *Medadhātu* manifest *Anyeddhuṣka*

Residing in *Asthi dhātu* manifest *Tṛtīyaka Jvara*

Residing in *Majjā dhātu* manifest *Caturthaka Jvara*

Residing in *Asthi-Majjā* manifest *Caturthaka Viparyaya Jvara*

सन्तत ज्वर (Santata Jvara)

स्त्रोतोभिर्विसृता दोषा गुरवो रसवाहिभिः ॥
सर्वदेहानुगाः स्तब्धा ज्वरं कुर्वन्ति सन्ततम् ।
दशाहं वा द्वादशाहं सप्ताहं वा सुदुःसहः ॥
स शीघ्रं शीघ्रकारित्वात् प्रशमं याति हन्ति वा ।
कालदूष्यप्रकृतिभिर्दोषस्तुल्यो हि सन्ततम् ॥
निष्प्रत्यनीकः कुरुते तस्माज्ज्ञेयः सुदुःसहः ।
यथा धातूंस्तथा मूत्रं पुरीषं चानिलादयः ॥
युगपच्चानुपद्यन्ते नियमात् सन्तते ज्वरे ।
स शुद्ध्या वाऽप्यशुद्ध्या वा रसादीनामशेषतः ॥

सप्ताहादिषु कालेषु प्रशमं याति हन्ति वा।
यदा तु नातिशुध्यन्ति न वा शुध्यन्ति सर्वशः॥
द्वादशैते समुद्दिष्टाः सन्ततस्याश्रयास्तदा।
विसर्गं द्वादशे कृत्वा दिवसेऽव्यक्तलक्षणम् ॥
दुर्लभोपशमः कालं दीर्घमप्यनुवर्तते ।
इति बुद्ध्वा ज्वरं वैद्य उपक्रामेत्तु सन्ततम्॥
क्रियाक्रमविधौ युक्तः प्रायः प्रागपतर्पणैः।

(C.S.Ci. 3/53-61)

सप्ताहं वा दशाहं वा द्वादशाहमथापि वा।
सन्तत्या योऽविसर्गी स्यात्सन्ततः स निगद्यते॥

(S.S.Utt. 39/69)

The heavy *Doṣās* pervaded the entire body through *Rasavaha Srotas* wherever they get stagnated produces *Santata Jvara.* Being acute onset either it gets cured (if *Doṣapāka* happens) or kills the patient (if *Dhātupāka* manifest) on 7th, 10th or 12th days. It produces extremely unbearable difficult condition. *Doṣās* involved in the development of this disease is similar in respect of *Kāla* (time), *Dūṣya (Dhātu, Mala), Prakṛti* (constitution) and having no counteracting factor causes *Satata Jvara,* which is quite unbearable. In this disease simultaneously *Dhātus, Mūtra, Puriṣa, Vāta* are afflicted as a rule. If *Rasadhātu* is not purified then patient dies on 7th, 10th or 12th days. Death is certain if *Rasadhātu* is partially purified.

Santata type of *Jvara* gets lodged in the twelve entities i:e seven *Dhātus,* three *Doṣās,* two *Śārīrika malās (Mūtra & Purīṣa).* Symptoms persist for longer duration even after pacification of symptoms on 12th day and it is very difficult for the management. Considering all above factors into consideration physician should initiate the treatment. Various treatment modalities are to be planned, but generally these therapies must be preceded by *Apatarpaṇa Cikitsā.*

Continous *Jvara* persisting for 7, 10 or 12th day without remission is the cardinal feature of *Santata Jvara.*

सततक ज्वर (Satataka Jvara)

रक्तधात्वाश्रयः प्रायो दोषं सततकं ज्वरम्॥
सप्रत्यनीकः कुरुते कालवृद्धिक्षयात्मकम्।
अहोरात्रे सततको द्वौ कालावनुवर्तते॥

(C.S.Ci 3/61-62)

Satataka Jvara manifest if the *Doṣās* gets lodged in *Raktadhātu* and it can be counteracted. Depending on nature of time bouts *Jvara* rises and falls. *Jvara* rises twice in twenty four hours.

अन्येद्युष्क ज्वर (Anyeddhuṣka Jvara)

कालप्रकृतिदूष्याणां प्राप्यैवान्यतमाद्बलम्।
अन्येद्युष्कं ज्वरं दोषो रुद्ध्वा मेदोवहाः सिराः ॥
सप्रत्यनीको जनयत्येककालमहर्निशि।

(C.S.Ci 3/63-64)

Anyeddhuṣka Jvara is caused by *Doṣa* which gets support of the strength either from time, constitution and *Dūṣyās* and produces obstruction in the *Medovaha Srotas.* It can be counteracted and *Jvara* rises once in 24 hours.

तृतीयक एवं चतुर्थक ज्वर (Tṛtīyaka evam Caturthaka Jvara)

दोषोऽस्थिमज्जगः कुर्यात्तृतीयकचतुर्थकौ ॥

(C.S.Ci 3/64)

तृतीयकस्तृतीयेऽह्नि चतुर्थेऽह्नि चतुर्थकः ॥

(S.S.Utt 39/70)

If *Doṣās* lodge in *Asthi dhātu* and *Majjādhātu* causes *Tṛtīyaka* and *Caturthaka Jvara* respectively. *Tṛtīyaka Jvara* appears on the third day and *Caturthaka Jvara* appears on the fourth day.

Some people says that *Anyeduṣka Jvara* manifest if *Doṣās* gets lodged in *Raktadhātu. Tṛtīyaka Jvara* manifest if *Doṣās* gets lodged in *Māmsa dhātu.* When the *Doṣās* gets lodged in *Medodhātu* it manifest *Caturthaka Jvara. (C.S.Ci. 3/65-66).*

Seed remains dormant in the soil until it gets favourable time and germinates after getting favourable time. In the same way *Doṣās* remain dormant in the *Dhātus* and get vitiated in appropriate time. *Tṛtīyaka* and *Caturthaka Jvara* manifest when the *Doṣas* gain strength in appropriate time and when the power of inhibiting factors is subsided.

Aggravated *Doṣās* in the opportunate time manifest *Jvara* and lose their strength and get located in their respective places. *Doṣās* again aggravated after getting favourable condition, as a result person will be afflicted with *Jvara*. (C.S.Ci. 3/68-70).

Sub classification of *Tṛtīyaka Jvara*

कफपित्तात्त्रिकग्राही पृष्ठाद्वातकफात्मकः ।
वातपित्ताच्छिरोग्राही त्रिविधः स्यात्तृतीयकः ॥
(C.S.Ci 3/71)

***Tṛtīyaka Jvara* is subclassified into 3 varieties.**

1. If *Tṛtīyaka Jvara* manifest due to aggravation of *Kapha* and *Pitta*, it is characterised by pain in lumbo-sacral region.
2. If *Tṛtīyaka Jvara* manifest due to predominance of *Vāta* and *Kapha*, it afflicts the back.
3. If *Tṛtīyaka Jvara* manifest due to aggravation of *Vāta* and *Pitta*, it afflicts the head.

Sub classification of *Caturthaka Jvara*

चतुर्थको दर्शयति प्रभावं द्विविधं ज्वरः ।
जङ्घाभ्यां श्लैष्मिकः पूर्वं शिरस्तोऽनिलसंभवः ॥
(C.S.Ci 3/72)

1. Symptoms commences from calf region due to predominant vitiation of *Kapha*.
2. Commencement of symptoms from head due to predominant vitiation of *Vāta*.

चतुर्थक विपर्यय (Caturthaka Viparyaya)

विषमज्वर एवान्यश्चतुर्थकविपर्ययः ।
त्रिविधो धातुरेकैको द्विधातुस्थः करोति यम् ॥
(C.S.Ci 3/73)

It manifest due to vitiation of all the three *Doṣās* and these *Doṣās* gets lodged in *Asthi* and *Majjā Dhātu*. Here *Jvara* remains for two days at a stretch followed by remission for one day. There after *Jvara*, again reappear and remain for two days.

प्रायशः सन्निपातेन दृष्टः पञ्चविधो ज्वरः ।
सन्निपाते तु यो भूयान् स दोषः परिकीर्तितः ॥
(C.S.Ci 3/74)

All varieties of *Viṣamajvara* manifest due to vitiation of all the three *Doṣās*. However the predominant *Doṣās* act as a causative factors.

ऋत्वहोरात्रदोषणां मनसश्च बलाबलात् ।
कालमर्थवशाच्चैव ज्वरस्तं तं प्रपद्यते ॥
(C.S.Ci 3/75)

Occurrence of *Jvara* at a particular time depends on strength or weakness of seasons, day and night, *Doṣa* and *Manas;* it is also due to past deeds.

धातुगत ज्वर (Dhātugata Jvara)

रसधातुगत ज्वर (Rasadhātugata Jvara)

गुरुत्वं दैन्यमुद्वेगः सदनं छर्द्यरोचकौ ।
रसस्थिते बहिस्तापः साङ्गमर्दो विजृम्भणम् ॥
(C.S.Ci 3/76)

गुरुता हृदयोत्क्लेशः सदनं छर्द्यरोचकौ ।
रसस्थे तु ज्वरे लिङ्गं दैन्यं चास्योपजायते ॥
(S.S.Utt. 39/83)

Rasadhātugata Jvara is characterised by heaviness, miserable expression, agitation, general malaise, vomiting, anorexia, increased external temperature, bodyache, yawning and nausea.

रक्तधातुगत ज्वर (Raktadhātugata Jvara)

रक्तोष्णाः पिडकास्तृष्णा सरक्तं ष्ठीवनं मुहुः ।
दाहरागभ्रममदप्रलापा रक्तसंस्थिते ॥
(C.S.Ci. 3/77)

रक्तनिष्ठीवनं दाहः स्वेदश्छर्दनविभ्रमौ ।
प्रलापः पिटिका तृष्णा रक्तप्राप्ते ज्वरे नृणाम् ॥
(S.S.Utt. 39/84)

Raktadhātugata Jvara is characterised by reddish skin eruptions having burning sensation, thirst, frequent spitting of blood, burning sensation, discolouration, giddiness, intoxication, delirium, sweating and vomitting.

मांसधातुगत ज्वर (Māmsadhātugata Jvara)

अन्तर्दाहः सतृण्मोहः सग्लानिः सृष्टविद्कता ।
दौर्गन्ध्यं गात्रविक्षेपो ज्वरे मांसस्थिते भवेत् ॥
(C.S.Ci. 3/78)

पिण्डिकोद्वेष्टनं तृष्णा सृष्टमूत्रपुरीषता ।
ऊष्मान्तर्दाहविक्षेपौ ग्लानिः स्यान्मांसगे ज्वरे ॥
(S.S.Utt. 39/85)

Māmsadhātugata Jvara is characterised by burning sensation internally, thirst, unconsciousness, fatigue, foul smell shaking movements of the body, twisting pain in the calves, more elimination of urine and stool.

मेदधातु ज्वर (Medadhātugata Jvara)

स्वेदस्तीव्रा पिपासा च प्रलापो वम्यभीक्ष्णशः ।
स्वगन्धस्यासहत्वं च मेदःस्थे ग्लान्यरोचकौ ॥
(C.S.Ci. 3/79)

भृशं स्वेदस्तृषा मूर्च्छा प्रलापश्छर्दिरेव च ।
दौर्गन्ध्यारोचकौ ग्लानिर्मेदःस्थे चासहिष्णुता ॥
(S.S.Utt. 39/86)

Medadhātugata Jvara is characterised by profuse perspiration, thirst, delirium, frequent vomiting, intolerance to one's body smell, fatigue, anorexia, fainting and restlessness.

अस्थिधातुगत ज्वर (Asthidhātugata Jvara)

विरेकवमने चोभे सास्थिभेदं प्रकूजनम् ।
विक्षेपणं च गात्राणां श्वासश्चास्थिगते ज्वरे ॥
(C.S.Ci. 3/80)

भेदोऽऽस्थ्नां कुञ्चनं श्वासो विरेकश्छर्दिरेव च ।
विक्षेपणं च गात्राणामेतदस्थिगते ज्वरे ॥
(S.S.Utt. 39/87)

Asthidhātugata jvara is characterised by diarrhoea, vomiting, pain in bones, contractions of body parts, dyspnoea and movements of body parts (convulusions).

मज्जा धातुगत ज्वर (Majjādhātugata Jvara)

हिक्का श्वासस्तथा कासस्तमकसश्चातिदर्शनम् ।
मर्मच्छेदो वहिः शैत्यं दाहोऽन्तश्चैव मज्जगे ॥
(C.S.Ci. 3/81)

तमःप्रवेशनं हिक्का कासः शैत्यं वमिस्तथा ।
अन्तर्दाहो महाश्वासो मर्मच्छेदश्च मज्जगे ॥
(S.S.Utt. 39/88)

Majjādhātugata Jvara is characterised by hiccup, breathlessness, cough, feeling of darkness infront of the eyes, pain in vital organs, feeling cold externally and burning sensation internally and vomiting.

शुक्रधातुगत ज्वर (Śūkradhātugata Jvara)

शुक्रस्थानगतः शुक्रमोक्षं कृत्वा विनाश्य च ।
प्राणं वाय्वग्निसोमैश्च सार्धं गच्छत्यसौ विभुः ॥
(C.S.Ci. 3/82)

मरणं प्राप्नुयात्तत्र शुक्रस्थानगते ज्वरे ।
शेफसः स्तब्धता मोक्षः शुक्रस्य तु विशेषतः ॥
(S.Su.Vtt. 39/89)

Śukradhātugata Jvara is characterised by excess liberation of *Śukra* and destruction of *Śukra* resulting in the extinction of life along with *Vāta, Agni* and *Soma;* stiffness in penis and lastly death ensues.

Prognosis of Dhātugata Jvara

रसरक्ताश्रितः साध्यो मेदोमांसगतश्च यः ।
अस्थिमज्जगतः कृच्छ्रः शुक्रस्थो नैव सिद्ध्यति ॥
(C.S.Ci. 3/83)

दग्ध्वेन्धनं यथा वह्निर्धातून् हत्वा यथा विषम् ।
कृतकृत्यो व्रजेच्छान्तिं देहं हत्वा तथा ज्वरः ॥
(S.S.Utt. 39/90)

Rasadhātugata Jvara, Raktadhātugata Jvara are curable. *Māmsadhātugata Jvara, Medadhātugata Jvara, Asthi dhātugata Jvara* and *Majjādhātugata Jvara* are curable with difficulty. *Sukradhātugata Jvara* is incurable

Fire subsides after utilizing all the fuel and poison nullifies after destroying all *dhātus,* similarly *dhātugata Jvara* subsides after killing the individual.

Eight Varieties of Jvara

अथ खल्वष्टाभ्यः कारणेभ्यो ज्वरः संजायते मनुष्याणां; तद्यथा-वातात्, पित्तात्, कफात्, वातपित्ताभ्यां, वातकफाभ्यां, पित्तकफाभ्यां, वातपित्तकफेभ्यः, आगन्तोरष्टमात् कारणात् ॥
(C.S.Ni. 1/17)

Eight specific factors are responsible for the causation of *Jvara* are as follows:–

1. *Vātaja* 2. *Pittaja* 3. *Kaphaja* 4. *Vata-Pittaja* 5. *Vāta-Kaphaja* 6. *Kapha-Pittaja* 7. *Vāta-Pitta-Kaphaja* and 8. *Āgantuja.*

वातज ज्वर हेतु (Vātaja Jvara Hetu)

रूक्षलघुशीतवमनविरेचनास्थापनशिरोविरेचनातियोगव्यायामवेगसन्धारणानशनाभिघातव्यवायोद्वेगशोकशोणितातिषेकजागरणविषमशरीरन्यासेभ्योऽतिसेवितेभ्यो वायुः प्रकोपेमापद्यते॥ *(C.S.Ni. 1/19)*

***Vātaja Jvara* manifest due to following etiological factors:–** Excess consumption of rough, light & cold substances, excess administration of *Vamana, Virecana, Āsthāpana,* errhines, physical exercise in excess, suppression of natural urges, fasting, trauma, sexual intercourse, anxiety, excessive letting, vigil during night, and uneven postures.

वातज्वर संप्राप्ति (Vātajvara Samprāpti)

स यदा प्रकुपित्तः प्रविश्यामाशयमूष्मणा सह मिश्रीभूयाद्यमाहारपरिणामधातुं रसनामानमन्ववेत्य रसस्वेदवहानि स्रोतांसि पिधायाग्निमुपहत्य पक्तिस्थानादूष्माणं बहिर्निरस्य केवलं शरीरमनुप्रपद्यते, तदा ज्वरमभिनिर्वर्तयति॥

(C.S.Ni. 1/20)

Due to above etiological factors *Vāta* gets aggravated and enters the *Āmāśaya* and combines with *Agni,* accompanying the *Rasa,* which is the first product after transformation of food, blocks the channels of *Rasa* and *Sveda* and diminishes the functions of *Agni* and takes out the *Agni* from the site of digestion, and spreads to all over body resulting into increased body temperature.

वात ज्वर लक्षण (Vāta Jvara Lakṣaṇa)

तस्येमानि लिङ्गानि भवन्ति; तद्यथा-विषमारम्भविसर्गित्वम्, ऊष्मणो वैषम्यं, तीव्रतनुभावानवस्थानानि ज्वरस्य, जरणान्ते दिवसान्ते निशान्ते घर्मान्ते वा ज्वरस्याभ्यागमनमभिवृद्धिर्वा, विशेषेण परुषारुणवर्णत्वं नखनयनवदनमूत्रपुरीषत्वचामत्यर्थंक्लृप्तीभावश्च; अनेकविधोपमाश्चलाचलाश्च वेदनास्तेषां तेषामङ्गावयवानां तद्यथा-पादयोः सुप्तता, पिण्डिकयोरुद्वेष्टनं, जानुनोः केवलानां च सन्धीनां विश्लेषणम्, ऊर्वोः सादः, कटीपार्श्वपृष्ठस्कन्धबाह्वंसोरसां च भग्नरुग्णमृदितमथितचटितावपाटितावनुन्नत्वमिव, हन्वोश्चाप्रसिद्धिः, स्वनश्च कर्णयोः शङ्खयोर्निस्तोदः, कषायास्यता आस्यवैरस्यं वा, मुखतालुकण्ठशोषः, पिपासा, हृदयग्रहः, शुष्कच्छर्दिः, शुष्ककासः, क्षवथूद्गारविनिग्रहः, अन्नरसखेदः, प्रसेकारोचकाविपाकाः, विषादजृम्भाविनाम- वेपथुश्रमभ्रमप्रलापप्रजागर रोमहर्षदन्तहर्षाः, उष्णाभिप्रायता, निदानोक्तानामनुपशयो विपरीतोपशयश्चेति वातज्वरस्य लिङ्गानि भवन्ति॥ *(C.S.Ci. 3/21)*

Characteristic features of *Vātaja Jvara* are as follows:–

- Irregularity in onset and remission
- Irregular temperature
- Irregularity in acuteness and mildness of temperature
- Emergence or aggravations of *Jvara* at the end of digestion, day, night and summer
- Excessive coarseness and redness of nail, eyes, face, urine, stool and skin.
- Various types of localised or shifting pain in different body parts like numbness in feet and cramps in calf, laxity in knee joint or in all joints. Debility in thigh; breaking, bursting, pressing, churning, cracking, extracting, and twisting pain in waist, sides, back, shoulders, arms, scapular region and chest respectively. Loss of functions of jaw; tinnitus.
- Piercing pain in temple region.
- Astringent taste or loss of taste in mouth.
- Dryness of mouth, palate and throat.
- Thirst, stiffness in heart, dry vomiting, dry cough, suppression of sneezing and eructations.
- Aversion to food & taste, salivation, anorexia, indigestion, depression, yawning, blending, tremors, exhaustion, giddiness, delirium, sleeplessness, horripilations, sensation in teeth.
- Liking for hot substances.
- Aggravation of the disease if patient again indulges etiological factors.
- Symptoms subside if patient consumes such things which are opposite to etiological factors.

पित्तज्वर हेतु (Pittajvara Hetu)

उष्णाम्ललवणक्षारकटुकाजीर्णभोजनेभ्योऽतिसेवितेभ्यस्तथा तीक्ष्णा- तपाग्निसंतापश्रमक्रोधविषमाहारेभ्यश्च पित्तं प्रकोपमापद्यते ॥ *(C.S.Ni. 1/22)*

The following etiological factors are responsible for the development of *Pitta Jvara*:–

- Excessive consumption of hot, sour, saline, alkaline and pungent food.
- Consumption of meals during indigestion.
- Excess exposure to intense sun & fire.
- Exhaustion, anger, and irregularity in meals.

पित्तज्वर संप्राप्ति (Pittajvara Samprāpti)

तद्यथा प्रकुपित्तमामाशयादूष्माणमुपसृज्याद्यमाहारपरिणामधातुं रसनामानमन्ववेत्य रसस्वेदवहानि स्रोतांसि पिधाय द्रवत्वादग्निमुपहत्य पक्तिस्थानादूष्माणं बहिर्निरस्य प्रपीडयत् केवलं शरीरमनुप्रपद्यते, तदा ज्वरमभिनिर्वर्तयति ॥

(C.S.Ni. 1/23)

Aggravated *Pitta* due to above etiological factors enters *Āmāśaya* and combines with *agni*, accompanying the *Rasa*, which is the first product after transformation of food, blocks the channels of *Rasa* & *Sveda* and impairs the functions of *Agni* due to its liquidity and takes out the *Agni* from the site of digestion and pervaded to entire body leads to increased body temperature.

Nidān Sevana (Consumption of Etiological factors) and during respective time
↓
Aggravation of *Pitta doṣa*
↓
Enters the *Āmāśaya*
↓
Combines with *Agni* and *Rasa*
↓
Blocks the channels of *Rasa* and *Sveda*
↓
Impairs the functions of *Agni* due to its liquidity
↓
Expel out the *Agni* from the site of digestion i.e. *Āmāśaya*
↓
Spread to all over body
↓
Increased body temperature
↓
Pittaja Jvara

पित्तज्वर लक्षण (Pittajvara Lakṣaṇa)

तस्येमानि लिङ्गानि भवन्ति; तद्यथा-युगपदेव केवले शरीरे ज्वरस्याभ्यागमनमभिवृद्धिर्वा भुक्तस्य विदाहकाले मध्यन्दिनेऽर्धरात्रे शरदि वा विशेषेण, कटुकास्यता, घ्राणमुखकण्ठौष्ठतालुपाकः, तृष्णा, मदो, भ्रमो, मूर्च्छा, पित्तच्छर्दनम्, अतीसारः, अन्नद्वेषः, सदनं, खेदः, रक्तकोठाभिनिर्वृत्तिः शरीरे, हरित हारिद्रत्वं नखनयनवदनमूत्रपुरीषत्वचाम्, अत्यर्थ मूष्मणस्तीव्रभावः, आतिमात्रं दाहः, शीताभिप्रायता, निदानोक्तानुपशयो विपरीतोपशयश्चेति पित्तज्वरलिङ्गानि भवन्ति॥ *(C.S.Ni. 1/24)*

***Pittaja Jvara* is characterised by following features:–**

- Simultaneous onset or aggravation of *Jvara* especially during mid digestion of food, midday, mid night and autumn season.
- Pungent taste in mouth.
- Inflammation of nose, mouth, throat, lips, and palate.
- Thirst, intoxication, giddiness, fainting.
- Vomiting of bitter substances, diarrhoea, dislike for food, malaise, sorrow, delirium.
- Appearance of red patches in the body.
- Greenish or yellowish discolouration of nails, eyes, mouth, urine, stool and skin, hyperpyrexia, intense burning sensation and liking for cold substances.
- Symptoms aggravates if patient again indulges in the causative factors and subsides if person adopts opposite qualities to its etiological factors.

कफज्वर हेतु (Kaphajvara Hetu)

स्निग्धगुरुमधुरपिच्छिलशीताम्ललवणदिवास्वप्नहर्षाव्यायामेभ्योऽतिसेवितेभ्यः श्लेष्मा प्रकोपमापद्यते॥

(C.S.Ni. 1/25)

***Kapha Jvara* manifest due to following etioligical factors :** Excessive consumption of unctuous, heavy, sweet, slimy, cold, sour, saline food, day sleep, exhilaration and lack of physical exercise.

कफज्वर संप्राप्ति (Kaphajvara Samprāpti)

स यदा प्रकुपित्तः प्रविश्यामाशयमूष्मणा सह मिश्रीभूयाद्यमाहारपरिणामधातुं रसनामानमन्ववेत्य रसस्वेदवहानि स्त्रोतांसि पिधायाग्निमुपहत्य पक्तिस्थानादूष्माणं बहिर्निरस्य प्रपीडयन् केवलं शरीरमनुप्रपद्यते, तदा ज्वरमभिनिर्वर्तयति॥

(C.S.Ni. 1/26)

Aggravated *Kapha* due to above etiological factors enters *Āmāśaya* and combines with *Agni*, accompanying the *Rasa*, which is the first product after transformation of food, blocks the channels of *Rasa* and *Sveda* and impairs the functions of *Agni* and takes out the *Agni* from the site of digestion and pervades the entire body resulting into increased body temperature.*Nidān Sevana* (Consumption of Etiological factors) and during respective time
↓
Aggravation of *Kapha doṣa*
↓

Enters the *Āmāśaya*
↓
Combines with *Agni* and *Rasa*
↓
Blocks the channels of *Rasa* and *Sveda*
↓
Impairs the functions of *Agni*
↓
Expel out the *Agni* from the site of digestion i.e. *Āmāśaya*
↓
Spread to all over body
↓
Increased body temperature
↓
Kaphaja Jvara

कफज्वर लक्षण (Kaphajvara Lakṣaṇa)

तस्येमानि लिङ्गानि भवन्ति; तद्यथा-युगपदेव केवले शरीरे ज्वरस्याभ्यागमनमभिवृद्धिर्वा भुक्तमात्रे पूर्वाह्णे पूर्वरात्रे वसन्तकाले वा विशेषेण, गुरुगात्रत्वम्, अनन्नाभिलाषः, श्लेष्मप्रसेकः, मुखमाधुर्यं हृल्लासः, हृदयोपलेपः, स्तिमितत्वं, छर्दिः, मृद्वग्निता, निद्राधिक्यं, स्तम्भः, तन्द्रा, कासः, श्वासः, प्रतिश्यायः, शैत्यं, श्वैत्यं च नखनयनवदनमूत्रपुरीषत्वचाम्, अत्यर्थं च शीतेतपिडका भृशमङ्गेभ्य उत्तिष्ठन्ति, उष्णाभिप्रायता, निदानोक्तानुपशयो विपरीतोपशयश्च इति (श्लेष्मज्वरलिङ्गानि भवन्ति) ॥ *C.S.Ci. 1/27*

***Kapha Jvara* is characterised by following features :**

- Simultaneous onset or aggravation of *Jvara* especially immediately after meals, forenoon, early night and spring season.
- Heaviness, aversion to food, salivation, sweetness in mouth, nausea, *hṛdayopalepa*, timidity, vomiting, sluggish functions of digestive fire, excessive sleep, stiffness, drowsiness, cough, breathlessness and coryza, coldishness, whitish discolouration of nails, eyes, face, urine, stool and skin.
- Excessive appearance of urticarial rashes all over the body, liking for hot substances. Symptoms aggravates if patient further indulges in the causative factors.
- Symptoms pacifies after administration of things which are having qualities opposite to its etiological factors.

द्वन्द्वज्वर एवं सन्निपातज ज्वर हेतु (Dvandva Jvara evam Sannipātaja Jvara Hetu)

विषमाशनादनशनादन्नपरिवर्तादृतुव्यापत्तेरसात्म्यगन्धोपघ्राणाद्विषोपहतस्य चोदकस्योपयोगाद्गरेभ्यो गिरीणां चोपश्लेषात् स्नेहस्वेदवमनविरेचनास्थापनानुवासनशिरोविरेचनानामयथावत्प्रयोगात् मिथ्यासंसर्जनाद्वा स्त्रीणां च विषमप्रजननात् प्रजातानां च मिथ्योपचाराद् यथोक्तानां च हेतूनां मिश्रीभावाद्यथानिदानं द्वन्द्वानामन्यतमः सर्वे वा त्रयो दोषा युगपत् प्रकोपमापद्यन्ते, ते प्रकुपित्तास्तयैवानुपूर्व्या ज्वरमभिनिर्वर्तयन्ति॥ *C.S.Ni. 1/28*

The following etiological factors manifest either *Dvandvaja Jvara* (involvement of two *Doṣās*) or *Sannipātaja Jvara* (involvement of three *Doṣās*).

- Irregularity in diet pattern, fasting, changes in food habit without following dietetic regimen, seasonal influences.
- Inhalation of substances which yields unpleasant smell, intake of poisonous water and poisonous substances and habitation near mountains.
- Improper administration of oleation therapy, sudation, emesis, purgation, ununctous and unctuous enema and errhines.
- Improper adaptation of dietetics after biopurification of the body, abnormal labour and intake of unwholesome regimen after delivery.
- Simultaneous aggravation of either two *Doṣās* or three *Doṣās* leads to manifestation of either *Dvandvaja Jvara* or *Sannipātaja Jvara* respectively.

द्वन्द्वज ज्वर लक्षण (Dvandvaja Jvara Lakṣaṇa)

वात-पित्तज ज्वर (Vāta-Pittaja Jvara)

शिरोरुक् पर्वणां भेदो दाहो रोम्णां प्रहर्षणम् ।
कण्ठास्यशोषो वमथुस्तृष्णा मूर्च्छा भ्रमोऽरुचिः ॥
स्वप्ननाशोऽतिवाग्जृम्भा वातपित्तज्वराकृतिः ।
C.S.Ci. 3/85-86

Characteristic features of *Vāta-Pittaja Jvara* are as follows : Headache, pain in joints, burning sensation, horripilation, dryness of throat and mouth, vomiting, thirst, fainting, giddiness, anorexia, sleeplessness, delirium and yawning.

वात कफज ज्वर (Vāta-Kaphaja Jvara)

शीतको गौरवं तन्द्रा स्तैमित्यं पर्वणां च रुक् ॥
शिरोग्रह: प्रतिश्याय: कास: स्वेदाप्रवर्तनम् ।
सन्तापो मध्यवेगश्च वातश्लेष्मज्वराकृति: ॥

C.S.Ci. 7/86-87

Characteristic feature of *Vāta-Kaphaja Jvara* are as follows : Feeling of coldness, heaviness, drowsiness, timidity, pain in joints, *Śirograha*, coryza, cough, absence of perspiration and mild increase in temperature.

श्लेष्मपित्त ज्वर (Śleṣmapitta Jvara)

मुहुर्दाहो मुहु: शीतं स्वेद: स्तम्भ: मुहुर्मुहु: ।
मोह: कासोरुचिस्तृष्णा श्लेष्मपित्तप्रवर्तनम् ॥
लिप्ततिक्तास्यता तन्द्रा श्लेष्मपित्तज्वराकृति: ।

C.S.Ci. 3/88

Śleṣma Pitta Jvara is characterised by frequent alternate feeling of burning sensation and coldishness, arrest of sweating, unconsciousness oftenly, cough, anorexia, thirst, discharge of *Śleṣma (Rasadhātu mala)* and *Pitta (Rakta dhātu mala)*, adhesion and bitterness in oral cavity and drowsiness.

सन्निपात ज्वर (Sannipāta Jvara)

सन्निपातज्वरस्योर्ध्वं त्रयोदशविधस्य हि ।
प्राक्सूत्रितस्य वक्ष्यामि लक्षणं वै पृथक् पृथक् ॥
भ्रम: पिपासा दाहश्च गौरवं शिरसोऽतिरुक् ।
वातपित्तोल्बणे विद्याल्लिङ्गं मन्दकफे ज्वरे ॥
शैत्यं कासोऽरुचिस्तन्द्रापिपासादारुग्व्यथा: ।
वातश्लेष्मोल्बणे व्याधौ लिङ्गं पित्तावरे विदु: ॥
छर्दि: शैत्यं मुहुर्दाहस्तृष्णा मोहोऽस्थिवेदना ।
मन्दवाते व्यवस्यन्ति लिङ्गं पित्तकफोल्बणे ॥
सन्ध्यस्थिशिरस: शूलं प्रलापो गौरवं भ्रम् ।
वातोल्बणे स्याद् द्व्यनुगे तृष्णा कण्ठास्यशुष्कता ॥
रक्तविण्मूत्रता दाह: स्वेदस्तृड् बलसंक्षय: ।
मूर्च्छा चेति त्रिदोषे स्याल्लिङ्गं पित्ते गरीयसि ॥
आलस्यारुचिहृल्लासदाहवम्यरतिभ्रमै: ।
कफोल्बणं सन्निपातं तन्द्राकासेन चादिशेत् ॥
प्रतिश्या छर्दिरालस्यं तन्द्राऽरुच्यग्निमार्दवम् ।
हीनवाते पित्तमध्ये लिङ्गं श्लेष्माधिके मतम् ॥
हारिद्रमूत्रनेत्रत्वं दाहस्तृष्णा भ्रमोऽरुचि: ।
हीनवाते मध्यकफे लिङ्गं पित्ताधिके मतम् ॥
शिरोरुग्वेपथु: श्वास: प्रलापश्छर्द्यरोचकौ ।
हीनपित्ते मध्यकफे लिङ्गं स्यान्मारुताधिके ॥
शीतको गौरवं तन्द्रा प्रलापोऽस्थिशिरोऽतिरुक् ।
हीनपित्ते वातमध्ये लिङ्गं श्लेष्माधिके विदु: ॥
श्वास: कास: प्रतिश्यायो मुखशोषोऽतिपार्श्वरुक् ।
कफहीने पितमध्ये लिङ्गं वाताधिके मतम् ॥
वर्चोभेदोऽग्निदौर्बल्यं तृष्णा दाहोऽरुचिर्भ्रम: ।
कफहीने वातमध्ये लिङ्गं पित्ताधिके विदु: ॥
सन्निपातज्वरस्योर्ध्वमतो वक्ष्यामि लक्षणम् ।
क्षणे दाह: क्षणे शीतमस्थिसन्धिशिरोरुजा ॥
सास्रावे कलुषे रक्ते निभुग्ने चापि दर्शने ।
सस्वनौ सरुजौ कर्णौ कण्ठ: शूकैरिवावृत: ॥
तन्द्रा मोह: प्रलापश्च कास: श्वासोऽरुचिर्भ्रम: ।
परिदग्धा खरस्पर्शा जिह्वा स्रस्ताङ्गता परम् ॥
ष्ठीवनं रक्तपित्तस्य कफेनोन्मिश्रितस्य च ।
शिरसो लोठनं तृष्णा निद्रानाशो हृदि व्यथा ॥
स्वेदमूत्रपुरीषाणां चिराद्दर्शनमल्पश: ।
कृशत्वं नातिगात्राणां प्रततं कण्ठकूजनम् ॥
कोठानां श्यावरक्तानां मण्डलानां च दर्शनम् ।
मूकत्वं स्रोतसां पाको गुरुत्वमुदरस्य च ॥
चिरात् पाकश्च दोषाणां सन्निपातज्वराकृति: ।

C.S.Ci. 3/90-109

13 Varieties of *Sannipātaja Jvara* has been described based on the predominance of *Doṣās* are as follows:–

It is characterised by

1. वातपित्तोल्बण मन्द कफ ज्वर *(Vāta Pittolbaṇa Manda Kapha Jvara) :* It is characterised by giddiness, thirst, burning sensation, heaviness and excessive headache.
2. वातश्लेष्मोल्बण मन्द पित्त ज्वर *(Vāta Śleṣmolbaṇa Manda Pitta Jvara) :* It is characterised by coldness, cough, anorexia, drowsiness, thirst, burning sensation, pain and distress.
3. पित्तकफोल्बण मन्दवात ज्वर *(Pitta Kapholbaṇa Manda Vāta Jvara) :* It is characterised by vomiting, burning sensation and coldishness alternatively, thirst, unconsciousness and pain in bones.
4. वातोल्बण मन्दपित्तकफ ज्वर *(Vātolbaṇa Manda Pitta Kapha Jvara) :* It is charcterised by pain

in joints, bone and head; delirium, heaviness, giddiness, thirst, dryness of throat and mouth.

5. पित्तोल्बण मन्दवात कफ ज्वर *(Pittolbaṇa Manda Vāta Kapha Jvara)* : It is characteris-ed by reddish discolouration of stool and urine, burning sensation, sweating, thirst, loss of strength and fainting.
6. कफोल्बण मन्द वात पित्त ज्वर *(Kapholbaṇa Manda Vāta Pitta Jvara)* : It is characterised by laziness, anorexia, nausea, burning sensation, vomiting, restlessness, giddiness, drowsiness and cough.
7. श्लेष्मोल्बण मध्यपित्त मन्दवात ज्वर *(Śleṣmolbaṇa Madhyapitta Manda Vāta Jvara)* : It is characterised by coryza, vomiting, laziness, drowsiness, anorexia, and loss of function of *Agni.*
8. पित्तोल्बण मध्य कफ मन्दवात ज्वर *(Pittolbaṇa Madhya Kapha Manda Vāta Jvara)* : It is characterised by predominance of *Pitta* followed by moderate *Kapha* and mild *Vāta,* yellowishness of urine and eyes, burning sensation, thirst, giddiness and anorexia.
9. वातोल्बण मध्यकफ मन्दपित्त ज्वर *(Vātolbaṇa Madhyakapha Mandapitta Jvara)* : It is characterised by predominance of *Vāta* followed by moderate *Kapha* and mild *Pitta,* headache, tremors, breathlessness, delirium, vomiting and anorexia.
10. कफोल्बण मध्यवात मन्द पित्त ज्वर *(Kapholbaṇa Madhya Vāta Manda Pitta Jvara)* : It is characterised by predominant *Kapha* moderate *Vāta* and mild *Pitta,* coldishness, heaviness, drowsiness, delirium and excessive pain in bone and head.
11. वातोल्बण मध्यपित्त मन्द कफ ज्वर *(Vātolbaṇa Madhya Pitta Manda Kapha Jvara)* : It is characterised by predominance of *Vāta* followed by moderate *Pitta* and mild *Kapha,* breathlessness, cough, coryza, dryness of mouth and excessive chest pain.
12. पित्तोल्बण मध्यवात मन्द कफ ज्वर *(Pittolbaṇa Madhya Vāta Manda Kapha Jvara)* : It is characterised by predominance of *Pitta* followed by moderate *Vāta* and mild *Kapha.* diarrhoea, sluggish digestion, thirst, burning sensation, anorexia and giddiness.
13. त्रिदोषोल्बण सन्निपात ज्वर *(Tridoṣālbaṇa Sannipāta Jvara)* : It is characterised by predominance of all the three *Doṣās.* Alternate feeling of burning sensation and coldish sensation, pain in bones, joints and head; excessive lachrymation, cloudyness, redness and crookedness in eyes, tinnitus and pain in ears, discomfort in the throat as if it feels like sharp edged bristles, drowsiness, unconsciousness, delirium, cough, breathlessness, anorexia, giddiness, tongue becomes charred & roughened, laxity in all body parts, haemoptysis, movements of head from here and there, thirst, sleeplessness and pain in cardiac region, delayed and little elimination of sweat, urine and faeces; moderate emaciation, continuous rumbling sound in the throat, reddish and blackish skin eruptions, muteness, inflammation in *Srotas,* heaviness in abdomen and delayed metabolic transformation of *Doṣās.*

साध्यासाध्यता (Sādhyāsādhyatā)

दोषे विबद्धे नष्टेऽग्नौ सर्वसंपूर्णलक्षणः ।
सन्निपातज्वरोऽसाध्यः कृच्छ्रसाध्यस्त्वतोऽन्यथा ॥
C.S.Ci. 3/109-110

Sannipāta Jvara is incurable if there is non elimination of abnormal *Doṣās,* complete loss of digestion, appearance of all the signs and symptoms in entirety and full; otherwise it is difficult to cure.

आगन्तुज ज्वर (Āgantuja Jvara)

आगन्तुरष्टमो यस्तु स निर्दिष्टश्चतुर्विधः ।
अभिघाताभिषङ्गाभ्यामभिचारभिशापतः ॥
C.S.Ci. 3/111-112

Mainly four etiological factors are described for the manifestation of *Āgantuja Jvara (Jvara* due to external causes) are as follows :

- *Abhighātaja* – Injury
- *Abiṣaṅgaja* – Ill attachment
- *Abhicāraja* – Evil tantric rituals
- *Abhiśāpaja* – Curse.

अभिघातज ज्वर (Abhighātaja Jvara)

शस्त्रलोष्टकशाकाष्ठमुष्ट्यरत्नितलद्विजैः ॥
तद्विधैश्च हते गात्रे ज्वरः स्यादभिघातजः ।
तत्राभिघातजे वायुः प्रायो रक्तं प्रदूषयन् ॥
सव्यथाशोफवैवर्ण्यं करोति सरुजं ज्वरम् ।
C.S.Ci. 3/112-114

Abhighātaja Jvara manifest due to trauma of weapons, stone, hunter, wood stick, fist, slap or teeth or such other means.

Due to above etiological factors *Vāta* aggravates and vitiates blood resulting in the manifestation of *Jvara* associated with distress, swelling, discolouration and pain.

अभिषङ्गज ज्वर (Abhiṣaṅgaja Jvara)

कामशोकभयक्रोधैरभिषक्तस्य यो ज्वरः ॥
सोऽभिषङ्गाज्ज्वरो ज्ञेयो यश्च भूताभिषङ्गजः ।
कामशोकभयाद्वायुः, क्रोधात् पित्ते, त्रयो मलाः ।
भूताभिषङ्गात् कुप्यन्ति भूतसामान्यलक्षणाः ।
भूताधिकारे व्याख्यातं तदष्टविधलक्षणम् ॥
विषवृक्षानिलस्पर्शात्तथाऽन्यैर्विषसंभवैः ।
अभिषक्तस्य चाप्याहुर्ज्वरमेकेऽभिषङ्गजम् ॥
चिकित्सया विषघ्न्यैव स शमं लभते नरः ।
C.S.Ci. 3/114-118

Causative factors for the genesis of *Abhiṣaṅgaja Jvara* are passion, grief, fear, anger, affliction by *Bhūtās* (evil spirits). *Pitta* aggravates due to passion, grief and anger and evil spirit afflicts all the three *Doṣās* and manifest the symptomatology according to the parts involved. Eight varieties of *Bhūtās* like insanity caused by *Gods, Ṛṣis, Manes, Gandharva, Yakṣa, Demons, Brahma Rākṣasa* and *Piśāca* are described in detail (C.S.Ci. 9/20) along with symptomatology.

Some people consider contact with poisonous air of the toxic plants and such other toxins are responsible for the manifestation of *Abhiṣaṅgaja Jvara* and its gets subsided by the appropriate usage of antidotes of these poisons.

अभिचारज (Abhicāraja) and अभिशापज (Abhiśāpaja)

अभिचाराभिशापाभ्यां सिद्धानां यः प्रवर्तते ॥
सन्निपातज्वरो घोरः स विज्ञेयः सदुः सहः ।
सन्निपातज्वरस्योक्तं लिङ्गं यत्तस्य तत् स्मृतम् ॥
चित्तेन्द्रियशरीराणामर्तयोऽन्याश्च नैकशः ।
प्रयोगं त्वभिचारस्य दृष्ट्वा शापस्य चैव हि ॥
स्वयं श्रुत्वाऽनुमानेन लक्ष्यते प्रशमेन वा ।
वैविध्यादभिचारस्य शापस्य च तदात्मके वा ॥
यथाकर्मप्रयोगेण लक्षणं स्यात् पृथग्विधम् ।
ध्याननिःश्वासबहुलं लिङ्गं कामज्वरे स्मृतम् ॥
C.S.Ci. 3/118-124

Jvara caused by evil tantric rituals is called *Abhicāraja Jvara. Jvara* caused by cursing of the saints is called *Abhiśāpaja Jvara.* Both these conditions are caused by all the three *Doṣās* and manifest symptomatology of these two are like *Sannipātaja Jvara Lakṣaṇa* along with afflictions of mind, sense organs and body. Both these can be determined from either direct observation of the performance of evil tantric rituals or curse or hearing from others or inference or pacification of *Jvara* after conducting counteracting procedures. *Kāmaja Jvara* is characterised by concentration of mind & frequency in breathing. *Śokaja Jvara* is characterised by excessive cry. *Bhayaja Jvara* is characterised by constant apprehension. *Krodhaja Jvara* is characterised by wrathfulness. *Bhūtaja Jvara* is characterised by observance of superhuman manifestations. *Viṣaja Jvara* is characterised by fainting, unconsciousness, intoxication and *Glāni*. Some of these above *Jvara's* fever appear first and then the Signs & Symptoms of *Kāma* etc. are manifested & in some others it is vice versa.

अभिचाराभिशापाभ्यां मोहस्तृष्णा च जायते ॥
S.S.Utt. 39/79

Delusion and thirst are the main clinical features *Abhicāraja & Abhiśāpaja Jvara.*

विषकृत ज्वर (Viṣakṛta Jvara)

श्यावास्यता विषकृते दाहातीसारहृद्ग्रहाः ।
अभक्तरुक् पिपासा च तोदो मूर्च्छा बलक्षयः ॥
S.S.Utt. 39/76

Jvara due to *Viṣa* (poison) manifest bluish discolouration in the mouth, burning sensation, diarrhoea, catching pain in heart, dislike for food, thirst, pricking pain, fainting and loss of strength.

ओषधिगन्धज ज्वर (Oṣadhigandhaja Jvara)

ओषधिगन्धजे मूर्च्छा शिरोरुग् वमथुः क्षवः ॥

S.S.Utt. 39/77

It is characterised by fainting, headache, vomiting and sneezing.

कामज ज्वर (Kāmaja Jvara)

कामजे चित्तविभ्रंशस्तन्द्राऽऽलस्यमरोचकः ।
हृदये वेदना चास्य गात्रञ्च परिशुष्यति ॥

S.S.Utt. 39/78

Kāma Jvara is charactrerised by disorders of mind, stupor, lassitude, loss of appetite, pain in heart and emaciation of the body.

भयज ज्वर (Bhayaja Jvara)/शोकज ज्वर (Śokaja Jvara)/ कोपज ज्वर (Kopaja Jvara)

भयात् प्रलापः शोकाच्च भवेत् कोपाच्च वेपथुः ।

S.S.Utt. 39/79

Bhayaja Jvara & *Śokaja Jvara* is characterised by delirium. *Kopaja Jvara* is characterised by tremors.

भूताभिषङ्गज ज्वर (Bhūtābhiṣaṅgaja Jvara)

भूताभिषङ्गादुद्वेगहास्यकम्पनरोदनम् ॥ *S.S.Utt. 39/79*

Bhūtābhiṣaṅgaja Jvara is characterised by irritation, laughing, tremors and weeping.

Doṣa vitiation in Āgantuja Jvara

ते पूर्वं केवलाः पश्चान्निजैर्व्यामिश्रलक्षणाः ।
हेत्वौषधविशिष्टाश्च भवन्त्यागन्तवो ज्वराः ।

C.S.Ci. 3/128-129

Āgantuja Jvara are at first unstinted but later they get mixed up with the signs and symptoms of *Nija Jvara*. However, *Āgantuja Jvara* have their own independent etiological factors and therapeutics.

गम्भीर ज्वर (Gambhīra Jvara)

गम्भीरस्तु ज्वरो ज्ञेयो ह्यन्तर्दाहेन तृष्णया ।
आनद्धत्वेन चात्यर्थं श्वासकासोद्गमेन च ॥

S.S.Utt. 39/92-93

Gambhīra Jvara is characterised by burning sensation inside the body, thirst & flatulence, severe breathlessness and cough.

दाहशीतादि ज्वर (Dāhaśitādi Jvara)

त्वक्स्थौ श्लेष्मानिलौ शीतमादौ जनयतो ज्वरे ।
तयोः प्रशान्तयोः पित्तमन्ते दाहं करोति च ॥
करोत्यादौ तथा पित्तं त्वक्स्थं दाहमतीव च ।
प्रशान्ते कुरुतस्तस्मिञ्छीतमन्ते च तावपि ॥
द्वावेतौ दाहशीतादि ज्वरौ संसर्गजौ स्मृतौ ।
दाहपूर्वस्तयोः कष्टः कृच्छ्रसाध्यश्च स स्मृतः ॥

S.S.Utt. 39/59-61

Śitādi Jvara is characterised by *Jvara* commencing with cold due to aggravation of *Kapha* and *Vāta*. When *Pitta* gets aggravated followed by pacification of *Kapha* and *Vāta*, produces burning sensation at the end.

Dāhādi Jvara

When aggravated *Pitta* enters the skin it produces severe burning sensation initially followed by coldness after its pacification.

Dāhapūrva and *Śītapūrva Jvara* are produced by the combination of two *Doṣās*. *Dāha Pūrva Jvara* is very difficult to treat.

Special Kinds of Sannipāta Jvara

अभिन्यास/हतौजस ज्वर (Abhinyāsa/Hataujasa Jvara)

विशेषञ्चात्र मे शृणु ॥

नात्युष्णशीतोऽल्पसंज्ञो भ्रान्तप्रेक्षी हतस्वरः ।
खरजिह्वः शुष्ककण्ठः स्वेदविण्मूत्रवर्जितः ॥
सास्त्रो निर्भुग्नहृदयो भक्तद्वेषी हतप्रभः ।
श्वसन् निपतितः शेते प्रलापोपद्रवायुतः ॥
तमभिन्यासमित्याहुर्हतौजसमथापरे ।

S.S.Utt. 39/38-41

Abhinyāsa Jvara is characterised by neither very hot nor very cold, semiconsciousness, unsteady eyes, loss of voice, coarse tongue, dryness of throat, absence of sweat, faeces and urine; lachrymation, discomfort in chest, dislike for food, loss of complexion, breathlessness, falling from cot and lies on the floor and delirium. It is also called *Hataujasa Jvara*.

Prognosis of Sannipātaja Jvara

सन्निपातज्वरं कृच्छ्रमसाध्यमपरे विदुः ॥
निद्रोपेतमभिन्यासं क्षीणमेनं हतौजसम् ।

सन्यस्तगात्रं संन्यासं विद्यात्सर्वात्मके ज्वरे ॥
S.S.Utt. 39/41-42

Sannipātaja Jvara is very difficult for management; some people says it is incurable. Among *Sannipāta Jvarās, Abhinyāsa* should be understood when patient has more sleep; *Hataujasa* when there is loss of sleep and it is also called *Sannyāsa* when there is loss of consciousness.

ओजोनिरोधज ज्वर (**Ojonirodhaja Jvara**)

ओजो विस्रंसते यस्य पित्तानिलसमुच्छ्रयात्।
स गात्रस्तम्भशीताभ्यां शयनेप्सुरचेतनः ॥
अपि जाग्रत्स्वपञ् जन्तुस्तन्द्रालुश्च प्रलापवान्।
संहृष्टरोमा स्रस्ताङ्गो मन्दसन्तापवेदनः ॥
ओजोनिरोधजं तस्य जानीयात् कुशलो भिषक् ॥
सप्तमे दिवसे प्राप्ते दशमे द्वादशेऽपि वा।
पुनर्घोरतरो भूत्वा प्रशमं याति हन्ति वा ॥
S.S.Utt. 39/43-45

Displacement of *Ojas* due to aggravated *Pitta* and *Vāta* manifest *Ojonirodhaja Jvara*. It is characterised by rigidity of the body, coldness, always desire to sleep, loss of consciousness, awaken from sleep oftenly followed by sleep, stupor, delirium, horripilations, debility of the body, mild increase in temperature, mild discomfort. It greatly enhances its peak on reaching the seventh, tenth or 12th day or pacifies or kills on those respective days.

तरुण ज्वर (**Taruṇa Jvara**)

स्त्रोतसां संनिरुद्धत्वात् स्वेदं ना नाधिगच्छति ।
स्वस्थानात् प्रच्युते चाग्नौ प्रायशस्तरुणे ज्वरे।
C.S.Ci. 3/132-133

It is characterised by absence of perspiration due to blockage of channels and displacement of *Agni* from its normal site.

आम ज्वर (**Āma Jvara**)

अरुचिश्चाविपाकश्च गुरुत्वमुदरस्य च ॥
हृदवस्याविशुद्धिश्च तन्द्रा चालस्यमेव च।
ज्वरोऽविसर्गी बलवान् दोषाणामप्रवर्तनम् ॥
लालाप्रसेको हृल्लासः क्षुन्नाशो विरसं मुखम्।
स्तब्धसुप्तगुरुत्वं च गात्राणां बहुमूत्रता ॥
न विड् जीर्णा न च ग्लानिर्ज्वरस्यामस्य लक्षणम् ।
C.S.Ci. 3/134-137

It is characterised by anorexia, indigestion, heaviness in abdomen, impurities in the heart, drowsiness, laziness, continuous and high fever, non elimination of *Doṣās,* salivation, nausea, loss of appetite, distaste in mouth, stiffness, numbness, heaviness, polyurea, improper formation of stool and absence of emaciation.

पच्यमान ज्वर (**Pacyamāna Jvara**)

ज्वरवेगोऽधिकस्तृष्णा प्रलापः श्वसनं भ्रमः।
मलप्रवृत्तिरुत्क्लेशः पच्यमानस्य लक्षणम्।
C.S.Ci. 3/136-137

Pacyamāna Jvara is characterised by high fever, thirst, delirium, breathlessness, giddiness and elimination of stool and *Kapha.*

निराम ज्वर (**Nirāma Jvara**)

क्षुत् क्षामता लघुत्वं च गात्राणां ज्वरमार्दवम्।
दोषप्रवृत्तिरष्टाहो निरामज्वरलक्षणम्।
C.S.Ci. 3/137-138

It is characterised by appearance of appetite, lightness of the body, reduction in body temperature, normal elimination of waste products, and duration of the disease is eight days.

पुनरावर्तक ज्वर (**Punarāvartaka Jvara**)

असंजातबलो यस्तु ज्वरमुक्तो निषेवते।
वर्ज्यमेतन्नरस्तस्य पुनरावर्तते ज्वरः ॥
दुर्हृतेषु च दोषेषु यस्य वा विनिवर्तते ।
स्वल्पेनाप्यपचारेण तस्य व्यावर्तते पुनः ॥
चिरकालपरिक्लिष्टं दुर्बलं हीनतेजसम्।
अचिरेणैव कालेन स हन्ति पुनरागतः ॥
अथवाऽपि परीपाकं धातुष्वेव क्रमान्मलाः।
यान्ति ज्वरमकुर्वन्तस्ते तथाऽप्यपकुर्वते ॥
दीनतां श्वयथुं ग्लानिं पाण्डुतां नान्नकामताम्।
कण्डूरुत्कोठपिडकाः कुर्वन्त्यग्निं च ते मृदुम् ॥
एवमन्येऽपि च गदा व्यावर्तन्ते पुनर्गताः।
अनिर्घातेन दोषाणामल्पैरप्यहितैर्नृणाम् ॥
निवृत्तेऽपि ज्वरे तस्माद्यथावस्थं यथाबलम्।
यथाप्राणं हरेद्दोषं प्रयोगैर्वा शमं नयेत् ॥
मृदुभिः शोधनैः शुद्धिर्यापना बस्तयो हिताः।
हिताश्च लघवो यूषा जाङ्गलामिषजा रसाः ॥
C.S.Ci. 3/333-340

Punarāvartaka Jvara manifest if person consumes avoidable food and activities during convalescence stage. It may also relapse even with slight irregularity in management in case where *Jvara* comes down but *Doṣās* are not eliminated properly. *Jvara* is persisting for longer duration associated with severe debility and loss of essence leads to death. In some cases *Doṣās* undergo metabolic transformation in *Dhātus* leading to pacification of *Jvara* but it produces harmful effects to the body like uneasiness, swelling, fatigue, pallor, loss of desire to take food, itching, skin rashes, pupils and sluggishness of *Jaṭharāgni*.

In other diseases, where symptoms are pacified but *Doṣās* are not eliminated properly, disease may relapse if person adopts unwholesome regimen. That's why it is very much necessary to remove the *Doṣās* from the body either through elimination or pacification therapies according to stage and strength of *Doṣās*. For this purpose, bio-purification of body with mild medicaments, along with *Yāpana* basti, vegetable soup and meat soup of wild animals are advised.

प्रलेपक ज्वर (Pralepaka Jvara)

तथा प्रलेपको ज्ञेयः शोषिणां प्राणनाशनः।
दुश्चिकित्स्यतमो मन्दः सुकष्टो धातुशोषकृद्॥

S.S.Utt. 39/54

Pralepaka Jvara observed in cases of *Rājayakṣmā* and it kills them. It is very difficult for management associated with mildness and dryness of tissues.

विपर्यय ज्वर (Viparyaya Jvara)

कफस्थानेषु वा दोषस्तिष्ठन् द्वित्रिचतुर्षु वा।
विपर्य्ययाख्यान् कुरुते विषमान् कृच्छ्रसाधनान्॥

S.S.Utt. 39/55

Viparyaya Jvara manifest if *Doṣās* remain in the seats of *Kapha* characterised by intermittent fevers, appear once in three or four days and it is very difficult for management.

Signs & Symptoms before pacification of Jvara

धातून् प्रक्षोभयन् दोषो मोक्षकाले बलीयते।
तेन व्याकुलचित्तस्तु म्रियमाण इवेहते॥

S.S.Utt. 39/321

During pacification of *Jvara Doṣās* become powerful and make the disturbances in the *Dhātu* and become liquified. It produces suffering to the mind and patient feels as if he is going to die. Manifest symptoms like rumbling sound, vomiting, increased movements of limbs etc. (C.S.Ci 3/324-328)

ज्वर मोक्ष लक्षण (Jvara Mokṣa Lakṣaṇa)

लघुत्वं शिरसः स्वेदो मुखमापाण्डु पाकि च।
क्षवथुश्चान्नकाङ्क्षश्च ज्वरमुक्तस्य लक्षणम्॥

S.S.utt. 39/322

Relief from *Jvara* may be understood by observing following symptomatology:– Feeling of lightness in the head, sweating, free from pallor and stomatitis, sneezing and desire for food.

साध्यासाध्यता (Sādhyāsādhyatā)

बलवत्स्वल्पदोषेषु ज्वरः साध्योऽनुपद्रवः।
सर्वथा विकृतिज्ञाने प्रागसाध्य उदाहृतः॥

A.H.Ni. 2/53

Jvara is curable in persons who are strong, which has mild increase of *Doṣās*, not associated with secondary complications. It becomes incurable if all the symptoms appears.

Incurability of fever

हेतुभिर्बहुभिर्जातो बलिभिर्बहुलक्षणः॥
ज्वरः प्राणान्तकृद्यश्च शीघ्रमिन्द्रियनाशनः।
ज्वरः क्षीणस्य शूनस्य गम्भीरो दैर्घरात्रिकः॥
असाध्यो बलवान् यश्च केशसीमन्तकृज्जवरः।
गम्भीरस्तु ज्वरो ज्ञेयो ह्यन्तर्दाहेन तृष्णया॥
आनद्धत्वेन चात्यर्थं श्वासकासोद्गमेन च।
आरम्भाद्विषमो यस्तु यश्च वा दैर्घरात्रिकः॥
क्षीणस्य चातिरूक्षस्य गम्भीरो यस्य हन्ति तम्।
विसंज्ञस्ताम्यते यस्तु शेते निपतितोऽपि वा॥
शीतार्दितोऽन्तरुष्णश्च ज्वरेण म्रियते नरः।
यो हृष्टरोमा रक्ताक्षो हृदि संघातशूलवान्॥
वक्त्रेण चैवोच्छ्वसिति तं ज्वरो हन्ति मानवम्।
हिक्काश्वासतृषायुक्तं मूढं विभ्रान्तलोचनम्॥
सन्ततोच्छ्वासिनं क्षीणं नरं क्षपयति ज्वरः।
हतप्रभेन्द्रियं क्षीणमरोचकनिपीडितम्॥
गम्भीरतीक्ष्णवेगार्तं ज्वरितं परिवर्जयेत्।

M.Ni. 2/66-73

- The fever caused by the excess indulgence of all the aetiological factors associated with appearance of all the signs and symptoms, loss of functions sensory organs and multi-organ failure leading to death of the patient.
- The fever that affects the weak individual associated with oedema, deep seated, high grade fever extended to night and hair-parting indicates incurable type of fever.

गम्भीर ज्वर (Gambhīra Jvara)

Gambhīra Jvara is characterised by burning sensation inside the body, thirst & obstruction to excreta, severe breathlessness and cough.

Incurable nature of jvara

- The fever which is irregular, perists all through the night, affects the weak strength individual associated with roughness and deep seated kills the patient.
- Complete loss of functions of sensory organs associated with frequent collapse and cold senation outside the body and burning sensation inside the body such patients will die definitely.
- If one suffers from horripilation/goose skin, redness in the eyes associated with severe pain in the heart and breathing through the mouth such patients will destroy and die soon.
- Development of emaciation due to fever associated with hiccup, dyspnoea, thirst, confused, one who rolls the eyes or casts side glances, troubled breathing associated with shallow intense breathing and weakness such patients die soon.
- One who lost his bodily lusture and loss of functions of sensory organs associated with emaciation, distaste/anorexia, hyperpyrexia (deep seated and high grade fever) such patients should not be considered for the treatment and should be rejected. 66-73 (Su. u. 39)

Signs & Symptoms before pacification of Jvara

दाहः स्वेदो भ्रमस्तृष्णा कम्पविड्भिदसंज्ञता ।
कूजनं चास्यवैगन्ध्यमाकृतिर्ज्वरमोक्षणे ॥
M.Ni. 2/74

Signs & Symptoms before pacification of Jvara are burning sensation, sweating, giddiness, thirst, trembling, diarrhoea, loss of sensation, quavering/uttering of any inarticulate sound and foul-smelling of the body.

Signs and symptoms on relief of fever

स्वेदो लघुत्वं शिरसः कण्डूः पाको मुखस्य च ।
क्षवथुश्चान्नलिप्सा च ज्वरमुक्तस्य लक्षणम् ॥
(M.Ni. 2/75)

Relief from Jvara may be understood by observing following symptomatology:

Sweating, feeling of lightness in the head, itching, stomatitis, sneezing, white coloration and ulceration in mouth, sneezing and desire for intake of food indicates the relief of fever from the pateint.

Prognosis of Jvara

प्रसक्तश्चाभिघातोत्थश्चेतनाप्रभवस्तु यः ॥ *S.S.Utt. 39/62*

- Persistence of *Jvara* for longer duration.
- *Jvara* produced by *Trauma* and that produced by disorders of mind are very difficult for management.

उपशय/पथ्य (Upaśaya/Pathya) (C.S.Ci. 3rd chapter) (S.S. Utt. 39th chapter)

- Laṅghana
- *Doṣa pācana* in case of *Taruṇa Jvara*
- *Ṣaḍaṅga Pānīya*
- *Vamana Karma*
- *Tarpaṇa*
- *Soup of Mudga or meat of wild animals*
- *Sarpipāna, Paya (milk)*
- *Virecana, Niruha, Anuvāsana, Mūrdha Virecana.*
- *Abhyaṅga, Pradeha, Pariṣeka* and *Avagāhana*
- *Paṭola, Kulaka, Pāpacelika (Pāṭhā), Karkoṭaka, Kaṭhilla* vegetables are useful.
- *Gharmāmbu* (Luke warm water) and *Madhya* (Alcoholic drinks).

सर्वज्वरेषु सुलघु मात्रावद्धोजनं हितम् ।
S.S.Utt. 39/145

Jvara patients should eat light food in little quantity.

Kalyāṇaka ghṛta *Mahākalyāṇaka ghṛta*
Triphalādi ghṛta *Pañcagavya ghṛta*
Prahlādana Taila

अनुपशय/अपथ्य (Anupaśaya/Apathya) (CS.Ci. 3rd chapter) (S.S. Vtt. 39th chapter)

गुर्व्यभिष्यन्द्यकाले च ज्वरी नाद्यात्कथञ्चन। न तु
तस्याहितं भुक्तमायुषे वा सुखाय वा॥
S.S.utt. 39/148-149

Jvara patients should not consume foods which are not easily digestible, which creates more moisture inside, apart from the usual times of taking meals. Such kinds of food is not beneficial to life or to induce happiness.

गुरूष्णास्निग्धमधुरान् कषायांश्च नवज्वरे।
आहारान् दोषपक्त्यर्थं प्रायशः परिवर्जयेत्॥
C.S.Ci. 3/195-196

Navajvara patients should not take heavy, hot, unctuous, sweet and astringent foods to facilitate metabolic transformation of *Doṣās*.

नवज्वरे दिवास्वप्नस्नानाभ्यङ्गान्नमैथुनम्।
क्रोधप्रवातव्यायामान् कषायांश्च विवर्जयेत्॥
C.S.Ci. 3/138-139

Patients of *Navajvara* should avoid sleep during day time, bath, massage, heavy food, sexual intercourse, anger, exposure to wind, exercise & *Kaṣāya*.

सज्वरो ज्वरमुक्तश्च विदाहीनि गुरूणि च।
असात्म्यान्यन्नपानानि विरुद्धानि च वर्जयेत्॥
व्यवायमतिचेष्टाश्च स्नानमत्यशनानि च।
तथा ज्वरःशमं याति प्रशान्तो जायते न च॥
व्यायामं च व्यवायं च स्नानं चङ्क्रमणानि च।
ज्वरमुक्तो न सेवेत यावन्न बलवान् भवेत्॥
C.S.Ci. 3/330-332

The following foods and activities are not conducive to *Jvara* patients.

- Foods and drinks which induces burning sensation, heavy, unwholesome & mutual antagonistic.
- Activities like sexual intercourse, excessive exhaustion, bath in excess.
- Large amount of meal.

During convalescence one should not perform exercise, sexual intercourse, bath, brisk walk until he regains strength.

In the case of the *Vāta*-related fever, patient should consume pure *Ghṛta*. In *Pitta*-generated fever cases, one should go through *Virecana* (purgation) therapy. If the fever relates to *Kapha*, light vomitting is prescribed. In case two of the above causes are involved, treatment suggested in both the conditions is to be opted. For example, if the fever relates to both *Vāta* and *Pitta*, drinking of pure *Ghṛta* is to be followed by *Virecana* (purgation) as well. If all the three indispositions are manifested, all of the three kinds of treatment suggested here are to be carried through. (B.R. 5/3)

Intake of meals in initial stage of fever

In the early stage of fever, meal before noon is to be abstained from. Two meals a day is to be avoided. Eating during the evening hours and taking eatables heavy to digest are also to be abstained from. (B.R. 3/8)

Role of laṅgana in fever

The patient of fever should first of all fast. *Laṅgana* (fasting) is the best method to get cure of fever caused by reasons other than the following: tuberculosis, loss of vital fluids, caused by *Vāta*, fear, anger, sexual lust, grief and excessive labour. (B.R. 5/11)

Prohibiting laṅgana (fasting) :

However, the physical strength and condition of the patient must be kept in mind before he is subjected to *Laṅgana* (fasting). The fasting should not be taxing on his life itself. For, energy in body is the state of health. The fasting is opted only up to a degree when it does not harm the critical energy of the patient. (B.R. 5/14)

Patients unfit for laṅgana (fasting):

Laṅgana (fasting) is not to be opted by such patients who are young, old, pregnant, or otherwise weak in body and suffering from such disease as giddiness, edema of mouth and those who may not bear the urge of thirst and hunger. (B.R. 5/15)

Characteristics of proper laṅgana (fasting):

Now the description of a successful *Laṅgana* (fasting) is described. If one passes the wind, urine and stool on right time and in proper quantity, if the body feels light, if the heart is not heavy by feeling, stomach is belching properly, if throat, mouth and tongue of the patient is free of cough and saliva and he feels cheerful, if there is proper sweating, if one feels thirst and hunger on time and if there is no pain in the mind and body of the patient, he should understand that his *Laṅgana* (fasting) has been properly carried through. (B.R. 5/16-17)

Helpful meals in cases of fever

If one is ailing by a fever caused by excessive labour, *Laṅgana* (fasting) or due to *Vāta*, one should take rice along with soup of mutton. In *Kapha* related fever, one should take rice along with *Yūṣa* prepared from *Mudga* lentil and water in 1:18 ratio. This *Yūṣa* should be treated with a little sugar and be given along with rice to the patients suffering through fever due to *Pitta*. (B.R. 5/35)

Use of red variety of rice and the like in fever

Older rice, particularly the *Śāli* and *Ṣaṣṭhī* (grown in sixty days) varieties are good for making *Yavāgū*, *odana* and *Lājā*. (*Lājā* is a preparation based on roasted grains of paddy). All these three preparations are capable to cure one from fever-related diseases. (B.R. 1/36)

Use of the mudga based yūṣa in fever

Those having fever should when feeling hungry consume Yūṣa cooked along with water in 18:1 ratio along with pulses like *Mudga*, lentil, gram, *Kulattha* and *Mukuṣṭha*. (B.R. 5/37)

Intake of vegetables in fever

The veg-preparation of the leaves of *Paṭola*, aubergine, *Kulaka*, *Kāravellaka*, *Karkoṭaka*, *Parpaṭakam*, *Gojivhā*, radish and *Guḍūcī* are good in fever related cases. (B.R. 5/38)

Useful meals in cases of fever

Person indisposed by fever must take at least in small quantity meals as suggested above. If such patients take no meal, they can feel weakness and in extreme cases they can even die of this loss of strength. (B.R. 5/39)

If the patient develops a disinterest in taking prescribed medicines and meals due to repeated use of them or due to loss of taste, he should take help of other doses. He should prepare different things and try to assume that the prescribed meals are tasteful. This can help the patient regain interest in suggested eatable. (B.R. 5/40)

Dietary Regulations for the patients of Sannipāta

Tṛikanṭaka (i.e. *Gokṣura*, *Kañṭakārī*, *Durālabhā*) and *Gokṣura* are known as *Tṛikañṭakavācya* herbs. Take all these three herbs along with *Vakṣayamāṇa* (sited ahead) *Pañcamṣṭika* herbs including *Yava*, *Badarī* (jujbe fruit), *Kulattha*, *Mudgaparṇī* and *Āmalakī*. Prepare decoction of these materials and obtain *Yavāgu* and *Peya* from it. Serve this preparation to the patient of *Sannipāta*. This recipe should be given repeatedly to the patient unless he feels better. One can also obtain *Yavāgu* from the decoction of *Tṛikanṭaka* and give it to patient. (B.R. 5/325)

The patient of *Viṣama jvara* should take liquor and meat of chicken, partridge and deer. (B.R. 5/386)

The wise and insightful physician should allow the patient of fever to take only the light meat of deer (venison) and *Lava*. Many physicians do not find it proper to allow such patients to take flesh of chicken, peacock, partridge, duck or the *Krauncha* bird. However, if after *Laṅgana* (fasting) the patient's *Vāta Doṣa* is restored physicians can prescribe small doses of chicken after considering proper timing. (B.R. 5/1280-1282)

Wholesome Foods and Conducts in middle stage of fever

Patient suffering from fever should use during his illness in middle stage following edible and dietary items: *Śāli* (old rice), brinjal, *Śobhāñjana*, *Kāravellaka*, *Vetrāgra*, fruits matured during *Āṣāḍha* month, *Paṭola*, *Karkoṭaka*, *Mūlaka* (radish), *Pūtikā-Upodikā*, *Mudga*, *Kulattha*, soup or *Yūṣa* of these vegetable and pulse etc, *Pāṭhā*, *Amṛtā*, *Vāstuka*, *Taṇḍulyaka*, *jīvantī* (*Śāka* or cooked vegetable), *Kākamācī*, grapes, *Kapittha*, *Dāḍima*, *Vikankta* (matured fruits) and like compatible meals and drugs that are easily digestible and having light properties in digestion. (B.R. 5/1435-1437)

Wholesome Foods and Conducts in later stage of fever

Virecana, *Vamana*, collyrium, snuff or inhalation, medicated smoking, *Anuvāsana*, *Śirāvyadha*, *Samśamana*, ointmenting, massage, bath, cold treatment, meat of *Aina*, *Kuliṅga*, peacock, wood cutter, rabit, partridge, chicken, *Krauncha*, *Kuraṅga*, *Pṛṣata*, *Cakora*, *Kapinjala*, duck and *Kālapuccha* – and the like animals and birds, milk and *Ghṛtam* of cow and goat, *haraḍa* fruit, water of hilly streams, castor oil, white sandal, moon rays or moonlight and embracement of beloved lady. (B.R. 5/1438-1441)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

During the early stage of fever, the following acts are to be avoided: sleeping in daytime, bathing, oil-massage, sexual intercourse, anger; facing the easterly wind, physical exercises and taking (any kind of) decoction. (B.R. 5/4)

Measures to be avoided at initial stage of fever

The measures which are prohibited for a patient in early stage of fever includes the following: flowing stream of cold water on head, application of *Kalka* of sandal and *Aguru*, bathing, application of therapies like *Virecana* (purgation), sleeping during the day time, intercourse with females, taking heavy exercises, drinking cold water or washing the hands and feet with it, getting agitated, sleeping in the direction facing cold easterly wind, sitting idle, moving a lot and taking meals that are difficult to digest. Similarly, patients should avoid bathing as it may lead to consumption, vomiting, insanity, fainting, giddiness, excessive thirst and anorexia. (B.R. 5/9-10)

Intake of hot and normal water during fever

In the fever caused due to disordered *Vāta*, *Kapha* and by a combination of the two one has the symptoms of excessive thirst. In such situation the patient should take lukewarm water in a small quantity. In cases of fever caused due to excessive intake of liquor and in cases of fever caused due to abnormal *Pitta*, the patient should drink cold water treated through herbs of *Mustaparpaṭakādi* group of herbs. (B.R. 5/24)

Merits of Śṛītaśīta water

Śṛītaśīta water (warmed water that has been made to return to the atomospheric temperature) are capable to promote digestive power and to destroy fever. These waters clear the vital channels of the body by enlarging their mouths and provide strength to the body. It also develops taste of foods and causes the body to sweat properly. Therefore, the lukewarm and warm water are very helpful for health. (B.R. 5/25)

Intake of liquids while suffering from fever:

Take pepper and dried ginger and treat with them the above *Ṣaḍanga pānīya* water and boil them along with *Lājā* (parched paddy). This preparation should be taken as a drink by the patients of fever. This pacifies their hunger and also reduces the degree of fever. This preparation is easy to digest as well. If the patient of fever is feeling pain in head, in sides and in lower abdomen, he should drink water treated and decocted in above fashion using *Gokṣura* and *Kañṭakāri* herbs along with *Lājā* (parched paddy). This preparation should be used as *Peya* as it promotes the urination and sweating. It is easy to digest and is capable to remove fever, pains of the sides and other parts of body. (B.R. 5/29-30)

Unwholesome Food and Conduct in early stage of fever:

Person afflicted with *Navajvara* (fever in primary stage) should restrict following eatables and conduct: *Snāna*, *Virecana*, sexual intercourse, physical exercise or exertion, massage, day sleep, milk, *Ghṛtam*, pulse, meat or flesh, buttermilk, liquors, sweet and heavy articles, liquids, cereals, open air and anger. (B.R. 5/1434)

Unwholesome Food and Conduct in general fever

Such items include the following: *Adhivāsana karma*, garland of red coloured flowers, wearing red cloth, suppression of vomiting, tooth-brushing by plant twig, unsuitable or unrelishing diet or food consumption, incompatible food and drinks, edible or food articles causing burning sensation, food heavy in digestion, impure, infected and pathogenic water, *Kṣārāmla*, *Patraśāka*, uncomptiable and sprouted grains, *Naladambu* (water treated with *Uśīra*), betel leaves, *Lakuca* and *Kalinda fruits*, *Odimatsya* (specific variety of fish), leafy vegetable, paste of sesame seed, edible mushroom, *Piṣṭavikṛta* meals (e. g. deep fried nuggets) and *abhiṣyandi* edibles like yogurt etc. (B.R. 5/1442-1445).

MCQs

1. *Rakta-dhatvashraya Vishama Jwara* is
   a. *Satata* b. *Tritiyaka*
   c. *Chaturthaka* d. All the above

2. *Jwara* which is *dussadhya* is
   a. *Rasadhatugata Jwara*
   b. *Paittika Jwara*
   c. *Vaikruta Jwara*
   d. *Manasika Jwara*

3. *Vatika Jwara Purvarupa* is
   a. *Sheela-vaikrita* b. *Jrimbha-atyartha*
   c. *Atinidra* d. *Amla-preeti*

4. *Shwasa vireka Chardi-Vikshepanam Gatranam* are the features of
   a. *Rakta-gata Jwara* b. *Mamsa-gata Jwara*
   c. *Medo-gata Jwara* d. *Asthi-gata Jwara*

5. *Maha-shwaaa* is seen in
   a. *Rasa-gata Jwara* b. *Majja-gata Jwara*
   c. *Shukra-gata Jwara* d. *Raktagata Jwara*

6. *Ojonirodhaja is described under*
   a. *Jwara* b. *Rajayakshma*
   c. *Kshayaja Kasa* d. *Kamala*

7. *Raktashteevanam dahah sweda chardan vibhram are the features observed in*
   a. *Rakta-gata Jwara*
   b. *Mamsa-gata Jwara*
   c. *Medo-gata Jwara*
   d. *Asthi-gata Jwara*

8. *Chittavibhramsha tandra* are features of
   a. *Shaapaja Jwara*
   b. *Abhishangaja Jwara*
   c. *Bhayaja Jwara*
   d. *Kamaja Jwara*

9. *Kama shoka bhaya krodhairabhisaktasya are the causative factors mentioned under*
   a. *Abhicharaja*
   b. *Abhighataja jwara*
   c. *Abhishapaja*
   d. *Abhishangaja Jwara*

10. *Adhika trushna, Pralapa, Bhrama* etc are features of
    a. *Antarvegi Jwara*
    b. *Dhatugata Jwara*
    c. *Dhatuleena Lakshanaa*
    d. *Jwara-mukta Lakshanaa*

11. नित्यं मन्दज्वरोरूक्षः शूनकस्तेन सीदति । स्तब्धाङ्गः श्लेष्मभूयिष्ठो
    a. *Vatabalasaka jwara*
    b. *Kaphapittaja jwara*
    c. *Vata-Kapha jwara*
    d. *Tridosha*

12. *Pralepaka Jwara observed* in
    a. *Rajayakshma* b. *Kshayaja kasa*
    c. *Ojonirodhaja jwara* d. *Gambhir jwara*

13. Commencement of symptoms *from shiras due to predominant vitiation of—in Chatur-thaka Jwara*
    a. *Vata dosha* b. *Kapha dosha*
    c. *Pitta dosha* d. *Rakta*

14. *Kaphapitta trikagrahi* features observed in
    a. *Tritiyaka jwara*
    b. *Chaturthaka jwara*
    c. *Satata jwara*
    d. *Santata jwara*

15. *Doshasthanasamshraya* in *Triteeyaka Jwara* observed in
    a. *Asthi-Mamsa dhatu*
    b. *Meda-Rakta dhatu*
    c. *Shukra dhatu*
    d. *Meda dhatu*

16. सप्ताहं वा दशाहं वा द्वादशाहमथापि वा are the features mentioned in context to
    a. *Tritiyaka jwara*
    b. *Chaturthaka jwara*
    c. *Santata jwara* d. *Satata jwara*

17. सप्तमे दिवसे प्राप्ते दशमे द्वादशेऽपिवा । पुनर्घोरतरो भूत्वा प्रशमं याति हन्ति वा ॥ features observed in
    - a. *Ojonirodhaja jwara*
    - b. *Chaturthaka jwara*
    - c. *Santata jwara*
    - d. *Satata jwara*
18. *Natyushna sheeto alpa samjno* is cardinal feature of
    - a. *Abhinyasa Jwara* b. *Ojonirodhaja jwara*
    - c. *Sannipata Jwara* d. *Dahadi Jwara*
19. *Hatujasa* is the synonym of
    - a. *Abhinyasa jwara*
    - b. *Santata-Jwara*
    - c. *Satata jwara*
    - d. *Punaravartaka jwara*
20. Nidropeta feature observed in
    - a. *Santata-Jwara* b. *Satata jwara*
    - c. *Punaravartaka jwara*
    - d. *Abhinyasa Jwara*
21. *Murccha shirorug vamathu kshava* are the features observed in
    - a) *Abhinyasa jwara*
    - b) *Punaravartaka jwara*
    - c) *Oshadigandhaja jwara*
    - d) *Satata jwara*
22. *Vepathu is the characteristic feature observed in*
    - a) *Bhayaja jwara* b) *Shojkaja jwara*
    - c) *Kamaja jwara* d) *Kopaja jwara*
23. *Agantuja Jwara* is classified into ________ category
    - a) 6 b) 4
    - c) 8 d) 5
24. If patient sees dreams as if he is having friendship with dogs indicates *Arista* in reference to which disease
    - a. *Jwara* b. *Shosa*
    - c. *Unmada* d. *Apasmara*
25. *Jaranante divasantenishante gharmante va jwarasyabhyagaman mabhivriddhirva* features observed in
    - a. *Vata Jwara*
    - b. *Pitta Jwara*
    - c. *Sannipatika Jwara*
    - d. *Vata-pittaja Jwara*
26. *Parvabheda, Romaharsha, Jrimbha* are the *lakshana* of which type of *Jwara*
    - a. *Vata-Kaphaja* b. *Vata-Pittaja*
    - c. *Pitta-Kaphaja* d. *Sannipathika*
27. *Liptatikthasyatha* is the *lakshana* of
    - a. *Vata-KaphajaJwara*
    - b. *Vata-PittajaJwara*
    - c. *Pitta-KaphajaJwara*
    - d. *Sannipathika Jwara*
28. *Mahanidra diwa jagaranam nishi* features observed in
    - a. *Sannipatika Jwara*
    - b. *Aganthu Jwara*
    - c. *Kapha-Pittaja Jwara*
    - d. *Vata-Kaphaja Jwara*
29. *Pratyaham vardhate jwara* is seen in
    - a. *Abhighataja* b. *Abhishangaja*
    - c. *Abhisapa* d. *Abhichara*
30. ध्याननिः श्वासबहुलं लिङ्गमं features observed in
    - a. *Shaapaja Jwara*
    - b. *Abhishangaja Jwara*
    - c. *Bhayaja Jwara*
    - d. *Kamaja Jwara*

Answer

1.	a	2.	c	3.	b	4.	d	5.	b
6.	a	7.	a	8.	d	9.	d	10.	a
11.	a	12.	a	13.	a	14.	a	15.	a
16.	c	17.	a	18.	a	19.	a	20.	d
21.	c	22.	d	23.	b	24.	a	25.	a
26.	b	27.	c	28.	a	29.	d	30.	d

CHAPTER 18

Masurika and Romantika

मसूरिका (Masūrikā)

Introduction

Chicken Pox. The *Masūrikā* is a disease which is signified as appearence of eruptions resembling *masūra* (lentils) in shape and size. Over-indulgence in eatables which are pungent, sour, salty, alkaline and are incompatible in nature and excess in quantity, irregular eating habits and fasting, intake of contaminated beans, leafy vegetables, air and water as well as bad planetary influences on certain areas of the country and in the community of people and like factors generate an increase of the *doṣās* in the body which subsequently in association with bad blood generate the above eruptions.

मसूरिका निदान एवं संप्राप्ति (Masūrikā Nidāna Evam Samprāpti)

कट्वम्ललवणक्षारविरुद्धाध्यशनाशनैः ।
दुष्टनिष्पावशाकाद्यैः प्रदुष्टपवनोदकैः ॥
क्रूरग्रहेक्षणाच्चापि देशे दोषाः समुद्धताः ।
जनयन्ति शरीरेऽस्मिन् दुष्टरक्तेन सङ्गताः ॥
मसूराकृतिसंस्थानाः पिडकाः स्युर्मसूरिकाः ।
M.Ni. 54/1-3

Causative factors for the development of *Masūrikā* are as follows:– Due to excessive consumption of foods which are pungent, sour, alkaline; antagonistic foods & drinks; eating during indigestion; not eating food or less intake of food; regular consumption of contaminated *niṣpāva*, leafy vegetables, air & water; bad planetary influences of certain parts of the country.

मसूरिका संप्राप्ति (Masūrikā Samprāpti)

Due to above etiological factors aggravation of *Doṣās* takes place. These aggravated *Doṣās* combined with polluted blood causes the development of skin eruptions which resemble like *Masūra* in size & shape is known as *Masūrikā*.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Pitttādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Excacerbated *doṣās* vitiates *Rakta*
↓
Leading to development of skin eruptions
↓
Masūrikā
(Which resemble like *Masūra* in size & shape)

मसूरिका संप्राप्ति घटक (Samprāpti Ghaṭaka of Masūrikā)

Doṣa	: *Tridoṣaja*
Dūṣya	: *Dhātu*→ *Rakta, Rasa, Māmsa* *Upadhātu*→ *Tvacā* *Śārīrika Mala* → *Puriṣa* *Dhātumala* → *Pitta, Kapha, Lasikā*
Agni	: *Jaṭharāgni, dhātvāgni*
Agniduṣṭi	: *Mandāgni*
Āma	: *Agnijanya*
Srotas	: *Raktavaha, Rasavaha, Māmsavaha*
Srotoduṣṭi	: *Saṅga, Vimārgagamana*
Adhiṣṭhāna	: *Tvacā*
Udbhavasthāna	: *Āmāśayottha*
Sancārasthāna	: *Tvacā*
Vyaktasthāna	: *Tvacā*
Prabhāva	: *Rasagata (Tvag gata), Raktagata, Pittaja, Kaphaja & Kapha-*

pittaja are easily curable. *Vātaja*, *Vātapittaja*, *Kaphavātaja* are very difficult to cure. *Sannipātaja* are incurable.

Svabhāva : *Āśukāri*

Roga bheda : 8 types based on *doṣa* involvement
7 types based on *dhātu* involvement

Roga Mārga : *Bāhya*

पूर्वरूप (Pūrvarupa)

तासां पूर्वं ज्वरः कण्डूर्गात्रभङ्गोऽरतिर्भ्रमः ॥
त्वचि शोथः सवैवर्ण्यो नेत्ररागश्च जायते ।
M.Ni. 54/3-4

Premonitory signs & symptoms of Masūrikā are as follows:–Fever, itching, pain all over the body, restlessness, giddiness, swelling & discoloration of skin and reddishness of eyes.

मसूरिका भेद (Masūrikā bheda)

Classification of Masūrikā

1. *Vātaja Masūrikā*
2. *Pittaja Masūrikā*
3. *Raktaja Masūrikā*
4. *Kaphaja Masūrikā*
5. *Tridoṣaja Masūrikā (Carma Piḍakā)*
6. *Kapha-Pittaja Masūrikā (Romāntikā)*
7. *Vata-Pittaja Masūrikā*
8. *Kapha-Vātaja Masūrikā*

Another classification based on involvement of *dhātus*:–

1. *Tvak gata Masūrikā*
2. *Raktagata Masūrikā*
3. *Māmsagata Masūrikā*
4. *Medagata Masūrikā*
5. *Asthigata Masūrikā*
6. *Majjāgata Masūrikā*
7. *Śukragata Masūrikā*

वातज मसूरिका (Vātaja Masūrikā)

स्फोटाः श्यावारुणा रूक्षास्तीव्रवेदनयाऽन्विताः ॥
कठिनाश्चिरपाकाश्च भवन्त्यनिलसंभवाः ।
सन्ध्यस्थिपर्वणां भेदः कासः कम्पोऽरतिः क्लमः ॥
शोषस्ताल्वोष्ठजिह्वानां तृष्णा चारुचिसंयुता ।
M.Ni. 54/4-6

Vātaja Masūrikā is charcterised by skin eruptions which are black, rough, severe pain, hard, delayed suppuration, pain in joints, bones & small joints, cough, tremors, restlessness, exhaustion, dryness of the palate, lips & tongue; thirst and anorexia.

पित्तज मसूरिका (Pittaja Masūrikā)

रक्ताः पीतसिताः स्फोटाः सदाहास्तीव्रवेदनाः ॥
भवन्त्यचिरपाकाश्च पित्तकोपसमुद्भवाः ।
विड्भेदश्चाङ्गमर्दश्च दाहस्तृष्णाऽरुचिस्तथा ॥
मुखपाकोऽक्षिरागश्च ज्वरस्तीव्रः सुदारुणः ।
M.Ni. 54/6-8

Pittaja Masūrikā is charcterised by skin eruptions which are red, yellow, associated with severe burning sensation & pain, quick suppuration, diarrhoea, general malaise, burning sensation, thirst, loss of appetite, stomatitis, redness of eyes, severe fever & very difficult for management.

रक्तज मसूरिका (Raktaja Masūrikā)

रक्तजायां भवन्त्येते विकाराः पित्तलक्षणाः ॥
M.Ni. 54/8

Raktaja Masūrikā is characterised by signs & symptoms similar to those of *Pittaja Masūrikā* are seen.

कफज मसूरिका (Kaphaja Masūrikā)

कफप्रसेकः स्तैमित्यं शिरोरुग्गात्रगौरवम् ।
हृल्लासः सारुचिर्निद्रा तन्द्रालस्यसमन्विताः ॥
श्वेताः स्निग्धा भृशं स्थूलाः कण्ड्वरा मन्दवेदनाः ।
मसूरिकाः कफोत्थाश्च चिरपाकाः प्रकीर्तिताः ॥
M.Ni. 54/9-10

Kaphaja Masūrikā is characterised by *Kaphapraseka,* stiffness, headache, heaviness, nausea, loss of appetite, sleep, stupor, lassitude, associated with skin eruptions which are white, soft, big, itching, mild pain and suppurates slowly.

त्रिदोषज मसूरिका/चर्मपिडका (Tridoṣaja Masūrikā/Carmapiḍakā)

नीलाश्चिपिटविस्तीर्णा मध्ये निम्ना महारुजः ।
चिरपाकाः पूतिस्त्रावाः प्रभूताः सर्वदोषजाः ॥

कण्ठरोधारुचिस्तम्भप्रलापारतिसंयुताः ।
दुश्चिकित्स्याः समुद्दिष्टाः पिडकाश्चर्मसंज्ञिताः ॥
M.Ni. 54/11-12

Tridoṣaja Masūrika or *Carmapiḍakā* is characterised by skin eruptions which are blue, flat, broad, depressed at the centre, severe painful, suppurating slowly, exudes pus which is foul smelling & large in quantity; obstruction in the throat, loss of appetite, stiffness, delirium, & restlessness is known as *Carmapiḍakā* & manifest due to involvement of all the three *Doṣās* & difficult to treat.

कफपित्तज मसूरिका/रोमान्तिका (Kapha-Pittaja Masūrikā/Romāntikā)

Kaphapittaja Masūrikā or *Romāntikā* characterised by skin eruptions which are small, red at the roots of the hairs making slight elevation associated with cough, loss of appetite and fever.

धातुगत मसूरिका (Dhātugata Masūrika)

त्वक्गत मसूरिका (Tvakgata Masūrikā) or रसगत मसूरिका (Rasagata Masūrikā)

तोयबुद्बुदसङ्काशास्त्वग्गतास्तु मसूरिका।
स्वल्पदोषाः प्रजायन्ते भिन्नास्तोयं स्रवन्ति च॥
M.Ni. 54/14

Tvakgata Masūrikā is characterised by skin eruptions which resembles like bubbles of water, exudes thin watery fluid on pricking associated with signs & symptoms of *Doṣās* which are mild in nature.

रक्तज मसूरिका (Raktaja Masūrikā)

रक्तस्था लोहिताकाराः शीघ्रपाकास्तनुत्वचः।
साध्या नात्यर्थदुष्टाश्च भिन्ना रक्तं स्रवन्ति च॥
M.Ni. 54/15

Raktaja Masūrikā is charcterised by skin eruptions which are red, suppurates quickly, thin skin, bleeds quickly after pricking and it is very difficult to treat.

मांसज मसूरिका (Māmsaja Masūrikā)

मांसस्थाः कठिनाः स्निग्धाश्चिरपाका घनत्वचः।
गात्रशूलतृषाकण्डूज्वरारतिसमन्विताः ॥
M.Ni. 54/16

Māmsaja Masūrikā is characterised by skin eruptions which are hard, slimy, suppurates slowly, thick skin, associated with pain all over the body, thirst, itching, fever & restlessness.

मेदज मसूरिका (Medaja Masūrikā)

मेदोजा मण्डलाकारा मृदवः किंचिदुन्नताः।
घोरज्वरपरीताश्च स्थूलाः स्निग्धाः सवेदनाः॥
संमोहारतिसंतापाः कश्चिदाभ्यो विनिस्तरेत्।
M.Ni. 54/17-18

Medaja Masūrikā is characterised by skin eruptions which are round, soft, slightly elevated, big, slimy, painful, high grade fever, delusion, restlessness, distress & only few persons get rid off this condition.

अस्थिमज्जागत मसूरिका (Asthi-Majjāgata Masūrikā)

क्षुद्रा गात्रसमा रूक्षाश्चिपिटाः किंचिदुन्नताः॥
मज्जोत्था भृशसंमोहवेदनारतिसंयुताः।
छिन्दन्ति मर्मधामानि प्राणानाशु हरन्ति हि॥
भ्रमरेणेव विद्धानि कुर्वन्त्यस्थीनि सर्वतः।
M.Ni. 54/18-20

Asthi-Majjāgata Masūrikā is charcterised by skin eruptions which are small, of the same level of skin, rough, flat, slightly elevated, associated with severe delusion, pain, restlessness, cutting pain in vital organs, takes away the life and severe pain in the bones as though stung by bees.

शुक्रगत मसूरिका (Śukragata Masūrikā)

पक्वाभाः पिडकाः स्निग्धाः सूक्ष्माश्चात्यर्थवेदनाः॥
स्तैमित्यारतिसंमोहदाहोन्मादसमन्विताः ।
शुक्रजायां मसूर्यां तु लक्षणानि भवन्ति हि॥
निर्दिष्टं केवलं चिह्नं दृश्यते न तु जीवितम्।
M.Ni. 54/20-22

Śukragata Masūrikā is characterised by skin eruptions which are ripe, slimy, minute, severe pain associated with stiffness, restlessness, delusion, burning sensation, toxicity and patient death is certain.

Involvement of *Doṣās* in *Dhātugata Masūrikā.*

दोषमिश्रास्तु सप्तैता द्रष्टव्या दोषलक्षणैः॥
M.Ni. 54/22

In all seven varieties of *Masūrikā* i.e.

Dhātugata Masūrikā signs & symptoms of one or two or three *Doṣās* are to be seen.

साध्यासाध्याता (Sādhyāsādhyatā)

त्वग्गता रक्तजाश्चैव पित्तजाः श्लेष्मजास्तथा ।
श्लेष्मपित्तकृताश्चैव सुखसाध्या मसूरिकाः ॥
वातजा वातपित्तोत्थाः श्लेष्मवातकृताश्च याः ।
कृच्छ्रसाध्यतमास्तस्माद्यत्नादेता उपाचरेत् ॥
असाध्याः सन्निपातोत्थास्तासां वक्ष्यामि लक्षणम् ।
प्रवालसदृशाः कश्चित् काश्चिज्जम्बूफलोपमाः ॥
लोहजालसमाः काश्चिदतसीफलसन्निभाः ।
आसां बहुविधा वर्णा जायन्ते दोषभेदतः ॥

M.Ni. 54/23-26

Rasagata (Tvag gata), Raktagata, Pittaja, Kaphaja & Kapha-pittaja are easily curable. *Vātaja, Vātapittaja, Kaphavātaja* are very difficult to cure, these *Masūrikā* should be treated with great effort & care.

Sannipātaja are incurable. Signs & symptoms of *Sannipātaja* are development of skin eruptions which resemble either colour of coral beads or fruits of *Jambū* or like an iron seive or like fruits of *Atasi* and many such colours develops due to combination of *Doṣās*.

अरिष्ट लक्षण (Ariṣṭa Lakṣaṇa)

कासो हिक्का प्रमेहश्च ज्वरस्तीव्रः सुदारुणः ।
प्रलापश्चारतिर्मूर्च्छा तृष्णा दाहोऽतिघूर्णता ॥
मुखेन प्रस्त्रवेद्रक्तं तथा घ्राणेन चक्षुषा ।
कण्ठे घुर्घुरकं कृत्वा श्वसित्यत्यर्थवेदनम् ॥
मसूरिकाभिभूतस्य यस्यैतानि भिषग्वरैः ।
लक्षणानि च दृश्यन्ते न दद्यादत्र भेषजम् ॥
मसूरिकाभिभूतो यो भृशं घ्राणेन निःश्वसेत् ।
स भृशं त्यजति प्राणाँस्तृषार्तो वायुदूषितः ॥
मसूरिकान्ते शोथः स्यात् कूर्परे मणिबन्धके ।
तथांऽसफलके चापि दुश्चिकित्स्यः सुदारुणः ॥

M.Ni. 54/27-30

If following signs & symptoms observed in *Masūrika* patients, then it should not be considered for treatment.

Cough, hiccough, *Prameha*, high fever, delirium, restlessness, fainting, thirst, burning sensation, curvatures of the body, bleeding from mouth, nose & eyes; wheezing sound in the throat, breathes with great difficulty. One who is breathing only expiration through nose, thirst & observation of vitiated symptoms of *Vāta* are indicative of death of the patient. Profound swelling in the elbow, wrist & shoulder joints develop in the last stage of *Masūrikā* and it is not possible to treat.

उपशय/पथ्य (Upaśaya/Pathya)

If the eruptions had become quite mature one should not try to treat the same as they are dried by wind itself. If a drug is administered at this stage it may vitiate the *Vāta doṣa*. As a result the poisons of *Masūrikā* would circulate inside the body itself. Therefore, it is better to support the *Vāta doṣa* of the body. For this purpose, one should take the *Rasāyanas* and the nutritious meals. (B.R. 59/15)

The patient of *Masūrikā* first go through *Vamana* (emetic) therapy, *Virecana* (purgation) therapy, blood letting and the like. After that, he should be served with the following eatables: old rice of *Śāli* variety, grams, *Mudga* beans, lentil beans, barley, meat of *Pratuḍa* birds (those who take their meals through beaks like pigeon, sparrow, *Krouñca*, parrot and the like), *Paṭola*, *Kāravellaka*, fruits of rainy season, banana fruits, drumsticks, *Mātulunga* lemon, *Drākṣā*, pomegranate and the like nutritious items. To treat eyes one should wash them with water treated with the *Gavedhu* and *Madhuka* herbs, camphor powder and drops obtained from conch shells. When the eruptions are matured one should take pulse soup of *Mudga* beans, meat soup of wild animals, leaves of *Śāli* plant, *Ghṛtam*, leaves of *Nirguṇḍī* plant and the like. The patient should take smoke (*Dhūpana*) therapy by burning *Gomaya* and *Guggulu*. If the eruptions are containing pus the patient should rub his body with ash of burnt reeds. He should also coat his body with *Kalka* of dried *Nimba* leaves and *Haridrā*. One could even treat the eruptions in manner sores are treated. The patient could also take appropriate decisions as per the guidelines above. It helps to make one comfortable in that disease. (B.R. 59/68-71)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The patient of *Masūrikā* should avoid the following: Intercourse, *Svedana* (fomentation), excessive labour, getting angry, taking contaminated water and polluted air, suppression of natural urges and the like. He should also not take oily edibles, meals that are heavy to digest, bitter and citrus in taste. (B.R. 59/72-73)

रोमान्तिका (Romāntika)

रोमाकूपोन्नतिसमा रागिण्यः कफपित्तजाः ।
कासारोचकसम्युक्ता रोमान्त्यो ज्वरमूर्विकाः ॥

(M.Ni. Utta. 54/13)

Romantika is characterized by eruptions in the skin and it arises at the root of hair follicles with slight elevation associated with red in colour, cough and loss of appetite. It is caused due to *Kapha* and *Pitta doṣa*. The disease will be always preceded by fever.

मसूरिकायाः प्रकारं रोमान्तिकामाह-रोमकूपोन्नतिसमा इत्यादि । रागिण्य इति लोहिताः, ज्वरपूर्विका इति ज्वरपूर्वह्वपाः ॥ (मधुकोश व्याख्या)

According to *Madhukoṣa* commentary.

मसूरिकायाः प्रकारं रोमान्तिकामाहः- It is a variety of *masūrikā*

रोमकूपोन्नतिसमा इत्यादितः- Elevated eruptions at the root of hair follicle.

रागिण्य इति लोहिताः- Red in colour

ज्वरपूर्विका इति ज्वरपूर्वह्वपाः- Associated with manifesteation of *jvara* as *pūrvarupa*.

CHAPTER

19

Fever

Note

Classification, types and patterns of fever :

- Fevers can be arbitrary classified into acute, sub-acute and chronic fevers based on duration.
- Acute fevers (<7 days in duration) are characteristics of infectious diseases such as malaria and viral-related upper respiratory tract infection
- Sub acute fevers (usually not more than 2 weeks in duration) may be seen in cases of typhoid fever and intra-abdominal abscess, among others
- Chronic or persistent fevers (>2 weeks duration) are typical of chronic bacterial infections such as tuberculosis, viral infections like HIV, cancers and connective tissue diseases
- However, any cause of acute fever can become persistent or chronic if untreated.
- Based on the height of body temperature, fever can also be classified into low grade, moderate grade, high grade and hyperpyrexia
- The height of body temperature may have some diagnostic and prognostic implications.
- Some studies have attributed high grade fevers in infants to serious bacterial infections, although others have also shown that children with high fevers are at equally high risk for serious bacterial infections and for viral illness.

Normal and febrile body temperature ranges (rectal temperatures).

Body temperature	°C	°F
Normal	37-38	98.6-100.4
Mild/low grade fever	38.1-39	100.5-102.2
Moderate grade fever	39.1-40	102.2-104.0
High grade fever	40.1-41.1	104.1-106.0
Hyperpyrexia	>41.1	>106.0

Three major fever types

Three major fever types have been described including sustained/continuous fever, intermittent fever and remittent fever

Continuous or sustained fever

- Continuous or sustained fever is defined as fever that does not fluctuate more than about 1 °C (1.5 °F) during 24 h, but at no time touches normal.
- Continuous fevers are characteristics of lobar and gram-negative pneumonia, typhoid, acute bacterial meningitis, urinary tract infection, among others
- Fever characterised by slow stepwise temperature rise and a high plateau are classical of typhoid fever.
- Fever associated with relative bradycardia (temperature pulse dissociation or Faget's sign) is a feature of untreated typhoid, leishmaniasis, brucellosis, Legionnaire's disease and psittacosis, Yellow Fever, among others.

Intermittent fever

- Intermittent fever is defined as fever present only for several hours during the day
- This pattern of fever can be seen in malaria, pyogenic infections, tuberculosis (TB), schistosomiasis, lymphomas, leptospira, borrelia, kala-azar, or septicemia

- Sources of continuous, intermittent or transient bacteraemia may lead to continuous, intermittent or transient fevers respectively.
- In malaria, depending on the species of parasite, fever can occur with a periodicity of 24 h (quotidian-due to plasmodium falciparum), 48 h (tertian - plasmodium ovale and vivax), or 72 h (quartan - Plasmodium malariae).
- The Pel-Epstein's fever is an intermittent low grade fever characterized by 3-10 days of fever with subsequent a febrile periods of 3-10 days.
- It is thought to be a typical but rare manifestation of Hodgkin's lymphoma.

Remittent fever

- Remittent fever is defined as fever with daily fluctuations exceeding 2 ^{0}C but at no time touches normal.
- Remittent fevers are often associated with infectious diseases such as infective endocarditis, rickettsiae infections, brucellosis, among others.
- Relapsing fevers refer to those that are recurring and separated by periods with low-grade fever or no fever.
- Periodic or relapsing fevers are seen in malaria, lymphoma, borrelia, cyclic neutropenia, and rat-bite fever.
- Fever associated with night sweats has been described in infectious diseases such as TB, Nocardia, brucellosis, liver or lung abscess and sub-acute infective endocarditis, as well as in non-infectious diseases such as polyarteritis *nodoșa* and cancers such as lymphomas.

Fever is then divided into three main categories

- Fever with localized signs
- Fever without localized signs
- Persistent fever of unknown cause (PUO)

Thermoregulation

- In healthy individuals, body temperature varies in relation to several environmental and biological factors such as time of day, site of temperature measurements, level of physical activity, age, sex and race, among others.
- In spite of this variability, body temperature is tightly regulated within a fairly constant range-a thermal set point (recently renamed thermal balance point through the process of thermoregulation).
- The old view that temperature regulation functions as a unified control system with a single thermoregulatory centre has been rejected. Based on recent evidence, core temperature is regulated by various relatively independent thermoeffector loops, each having its own afferent and efferent branches.
- Hence, the regulation of body temperature is dependent on a thermoregulatory circuitry. However, the preoptic region of the anterior hypothalamus is still considered the major thermoregulatory centre in the CNS where peripheral and centrally generated temperature signals are received and integrated.
- The preoptic region consists of heat sensitive neurons, namely warm and cold sensitive neurons, which are activated or inhibited in response to temperature changes.
- In cold environments, decrease firing of warm sensitive neurons and increase firing of cold sensitive neurons leads to activation of heat gain mechanisms and prevention of heat loss (i.e. by skin vasoconstriction, piloerection, decrease sweating, increased muscle contraction, non-shivering thermogenesis, and seeking warm clothing or environments).
- In hot environments, increase firing of warm sensitive neurons and decrease firing of cold sensitive neurons leads to activation of heat loss mechanisms and inhibition of heat gain mechanisms (i.e. by sweating, removal of clothing or seeking cold environments).

- Ultimately, body temperature is kept within a normal regulated limit by a delicate balance between heat loss and heat gain.

Defining normal and febrile body temperatures

Fever: Temperature Measurement

- Rectal temperature measurement is considered to be the gold standard for children 3 years of age or younger. The most widely accepted definition of fever is rectal temperature of 38 ⁰C (100.4 ⁰F) or higher. It is important to consider that infants, especially those younger than 2 months of age, may have a blunted febrile (or hypothermic) response to infection. Hence, lack of fever should not be used as a criterion for ruling out infection in infants. Although rectal temperature measurement is the gold standard, it should be avoided in neutropenic immunocompromised patients, in whom rectal manipulation may seed the blood with bacteria.
- Oral thermometry can be considered for cooperative patients who are older than 4-5 years of age.
- Axillary temperatures are commonly done and tympanic membrane and temporal artery temperatures are newer modalities with some studies examining their reliability.
- Axillary temperatures are less precise than rectal temperatures. There is a correlation between axillary and rectal temperature measurements; the axillary temperature is usually 0.5-0.85 ⁰C lower.
- Tympanic membrane thermometers are often inaccurate in children. Temporal artery temperature measurement correlates well with rectal temperature in some studies, but has been shown to be inferior when patients are febrile. It can be considered in settings when children are not likely to be febrile and are over 3 months of age. When detection of fever is critical for diagnosis and management, rectal temperatures should be used in the child 3 years of age and younger.
- In the geriatric group (>65 years), who are likely to have lower body temperatures, IDSA defines fever as single oral temperature >100 ⁰F (>37.8 ⁰C) or repeated oral temperatures >99 ⁰F (>37.2 ⁰C) or rectal temperatures >99.5 ⁰F (>37.5 ⁰C) or an increase in temperature of >2 ⁰F (>1.1 ⁰C) over the baseline temperature.
- Fever has both positive and negative effects. High body temperatures may impair the reproduction and survival of some invading microorganisms by decreasing required nutrients, such as free iron, or by increasing immunologic responses such as phagocytosis.
- However, at extremely high temperatures, immunologic responses may be impaired. Fever increases the basal metabolic rate by 10-12% for each degree Celsius elevation of temperature. This increases oxygen consumption, carbon dioxide production, and fluid and caloric needs. Fluid requirements increase 100/ mL/m2/day for each 1⁰C rise in temperature above 37.8⁰C.
- Heat illness must be distinguished from fever as a cause for elevated body temperature.
- In heat illness, there is an unregulated rise in body temperature, despite the fact that the hypothalamic set point is normal. It can result from excessive heat production or inadequate heat dissipation.
- Temperatures may reach extreme heights and can result in multiorgan dysfunction and death. Hence restoration of normal body temperature in heat illness is mandatory.

Physical Examination

- Many children will have a source for fever identified on their history and/or physical examination.
- If no focus of infection is found on the physical examination, the clinician must rely

on history and observation to determine the appropriate next steps. The child may appear ill or well. Ill-appearing children are typically lethargic or irritable. They may show signs of shock, including weak peripheral pulses, tachycardia, poor perfusion, respiratory distress, mottling, cyanosis, or decreased mental status. After thorough clinical and laboratory evaluation, ill-appearing children should be admitted to the hospital, and will likely need empiric antibiotic treatment.

Causes of Hyperthermia

Excessive Heat Production

- Exertion
- Heat stroke (exertion)
- Malignant hyperthermia (anesthesia induced)
- Neuroleptic malignant syndrome
- Catatonia
- Tetanus
- Status epilepticus
- Delirium
- Endocrine disorders (hyperthyroidism, pheochromocytoma)
- Drugs (cocaine, amphetamines, ephedrine, phencyclidine, tricyclic antidepressants, lysergic acid diethylamide(LSD), lithium, thyroid hormone, salicylates)

Diminished Heat Dissipation

- Heat stroke
- Occlusive dressings
- Dehydration
- Extensive burns (including severe sunburn)
- Anhidrotic ectodermal dysplasias
- Anticholinergic-like drugs (atropine, antihistamines, phenothiazines, tricyclic antidepressants)
- Autonomic neuropathy
- Spinal cord level paralysis (spinal crisis)
- Possible overbundling (especially in a warm environment)
- Therapeutic hyperthermia

Hypothalamic Dysfunction (Usually associated with hypothermia)

- Stroke
- Encephalitis
- Granulomatous processes (sarcoid, tuberculosis, eosinophilic)
- Trauma
- Central: idiopathic
- Phenothiazines
- Hemorrhage

Etiology and pathogenesis of Jvara

मिथ्याहारविहारभ्यां दोपा ह्यामाशयाश्रयाः ।
बहिर्निरस्य कोष्ठाग्निं ज्वरदाः स्यू रसानुगाः ॥

The *Doṣas* aggravated in the *Āmāśaya* by the consumption of contrary foods and activities leading to the expulion of *Agni* from the *Āmāśaya* associated with *rasa dhātu* leading to developmentt of fever.

Note-

Causes of fever : Fever is the result of your immune system's response to a foreign invader. These foreign invaders include viruses, bacteria, fungi, drugs, or other toxins.

Fever : the role of pyrogens and cryogens

- The initiation, manifestations and regulation of the febrile response are dependent on the pyrogenic and anti-pyretic properties of various exogenous and endogenous substances.
- While pyrogens directly or indirectly lead to fever, cryogens prevent excessive temperature elevation. It is the balance in the interactions between pyrogens and cyrogens that determine the height and duration of the febrile response to any immune challenge.

Pyrogens

- The foreign invaders are considered fever-producing substances (called pyrogens), which trigger the body's immune response. Pyrogens signal the hypothalamus in the brain to increase the body temperature set point in order to help the body fight off the infection.

- Substances that cause fever are called pyrogens.
- Pyrogens can come from inside or outside the body.
- Microorganisms and the substances they produce (such as toxins) are examples of pyrogens formed outside the body.
- Pyrogens are classified into exogenous (produced outside the host) and endogenous (produced within the host) pyrogens based on their site of production.
- Pyrogens formed inside the body are usually produced by monocytes and macrophages (two types of white blood cells). Endogenous pyrogens are mainly pyrogenic cytokines including interleukins (IL) 6, IL-1, interferon gamma (INF-γ) and ciliary neurotropic factor (CNTF) and tumour necrosis factor (TNF α), among others.
- Pyrogens from outside the body can cause fever by stimulating the body to release its own pyrogens or by directly affecting the area of the brain that controls temperature. Exogenous pyrogens are, essentially, part or whole micro-organisms or products of micro-organisms such as toxins.
- Infection is not the only cause of fever.
- Fever may also result from inflammation, a reaction to a drug, an allergic reaction, autoimmune disorders (when the body produces abnormal antibodies that attack its own tissues), and undetected cancer (especially leukemia, lymphoma, or kidney cancer).

Cryogens

- Cyrogens include anti-inflammatory cytokines (e.g. IL-10), hormones (e.g. a-melanocyte stimulating hormone, corticotrophin and corticotrophin releasing hormone) and many other neuroendocrine products (e.g. neuropeptide Y, bombesin, and thyroliberin), cytochrome P-450 (P-450), among others.
- They exert their anti-pyretic effects by inhibiting synthesis of pyrogenic cytokines (e.g. glucocorticoids), cytokine receptors blockade (e.g. IL-1 receptor antagonist), and increasing heat loss by enhancing sensitivity of warm sensitive neurons (e.g. bombesin), among other mechanisms. These endogenous antipyretic systems protect the host against the destructive consequences of unchecked fever.

Many disorders can cause fever. They are broadly categorized as :

- Infectious (most common)
- Neoplastic (cancer)
- Inflammatory
- An infectious cause is highly likely in adults with a fever that lasts 4 days or less.
- A noninfectious cause is more likely to cause fever that lasts a long time or returns.

Many cancers cause fever

- Inflammatory disorders that cause fever include joint, connective tissue, and blood vessel disorders such as rheumatoid arthritis, systemic lupus erythematosus (lupus), and giant cell arteritis.
- Also, an isolated, short-lived (acute) fever in people with cancer or a known inflammatory disorder is most likely to have an infectious cause.
- In healthy people, an acute fever is unlikely to be the first sign of a chronic illness.
- Drugs sometimes cause fever. For example, beta-lactam antibiotics (such as penicillin) and sulfa drugs can trigger a fever.
- Drugs that can cause an extremely high temperature include certain illicit drugs (such as cocaine, amphetamines, or phencyclidine), anesthetics, and antipsychotic drugs.

Most common causes

- Virtually all infectious disorders can cause fever.
- But overall, the most likely infectious causes are
- Upper and lower respiratory tract infections
- Gastrointestinal infections
- Urinary tract infections (UTIs)

- Skin infections
- Most acute respiratory tract and gastrointestinal infections are viral.

Risk factors

- Certain conditions (risk factors) make people more likely to have a fever. These factors include the following:
- The person's health status
- The person's age
- Certain occupations
- Use of certain medical procedures and drugs
- Exposure to infections (for example, through travel or contact with infected people, animals, or insects)

Some Causes of Fever Based on Risk Factors

Risk Factor	Cause
None (healthy)	Upper or lower respiratory tract infection Gastrointestinal infection Urinary tract infection Skin infection
Hospitalization	Infection related to a catheter inserted in a vein (IV catheter infection) Urinary tract infection, particularly in people with a urinary catheter Pneumonia, particularly in people on a ventilator Atelectasis (collapse of part of a lung due to an airway blockage, rather than an infection) Infection or a pocket of blood (hematoma) at the site of surgery Deep vein thrombosis or pulmonary embolism Diarrhea (due to-Clostridioides difficileinduced colitis) Drugs Transfusion reaction Pressure sores
Travel to areas where an infection is common (endemic areas)	Malaria Viral hepatitis Disorders that cause diarrhea Typhoid fever Zika virus, chikungunya, Ebola virus, Japanese encephalitis, rabies, measles, and yellow fever Dengue fever Fungal infections such as coccidioidomycosis, blastomycosis, or histoplasmosis Rickettsial infections (such as African tick-bite fever and Mediterranean spotted fever)
Exposure to insects or animals that carry disease-causing organisms, called vectors (in the United States)	Ticks: Rickettsiosis, ehrlichiosis or anaplasmosis, Lyme disease, babesiosis, relapsing fever, or tularemia Mosquitoes: Encephalitis caused by arboviruses, malaria, or Zika virus infection Wild animals: Tularemia, rabies, or hantavirus infection Fleas: Plague Domestic animals: Brucellosis, cat-scratch disease, Q fever, or toxoplasmosis Birds: Psittacosis Reptiles: Salmonella infection Bats: Rabies or histoplasmosis

A weakened immune system (immunocompromise)	Viruses: Varicella-zoster virus infection or cytomegalovirus infection Bacteria: Infection due to pneumococci, meningococci, Staphylococcus aureus, Pseudomonas aeruginosa, Nocardia, or mycobacteria Fungi: Infection due to Candida, Aspergillus, Histoplasma, Coccidioides, microsporidia, Pneumocystis jirovecii, or fungi that cause mucormycosis Parasites: Infection due to Toxoplasma gondii, Strongyloides stercoralis, Cryptosporidium, or Cystoisospora belli
Drugs that can increase heat production	Amphetamines Cocaine Phencyclidine (PCP) Methylenedioxymethamphetamine (MDMA, or Ecstasy) Antipsychotic drugs Anesthetics
Drugs that can trigger fever	Beta-lactam antibiotics (such as penicillin) Sulfa drugs Phenytoin Carbamazepine Procainamide Quinidine Amphotericin B Interferons (drugs that are based on substances produced by the immune system and that help block the reproduction of viruses)

Fever of unknown origin (FUO)

A fever of unknown origin may be diagnosed when :

- People have a fever of at least 101 ^{0}F (38.3 ^{0}C) for several weeks
- Extensive investigation does not detect a cause

Pathophysiology of the febrile response

- The development of the febrile response is akin to the normal thermoregulatory processes that follow exposure to cold temperatures. However, in fever the thermal balance point is reset to a higher level such that normal peripheral and central body temperatures are now sensed as cold temperature signals by the thermoregulatory circuitry.
- Consequently, fever is different from heat stroke and hyperthermia where body temperature is elevated without a corresponding elevation of the thermal balance point.
- Like thermoregulation, evolving evidence suggest that the generation of fever follows multiple independent afferent and efferent mechanisms depending on the site, nature and severity of inflammation.
- The various biological molecules involved in the generation of the febrile response and the pathways implicated in these responses are discussed in the following section.

The fever pathways

Fever signals carried by exogenous and endogenous pyrogens ultimately lead to reset of the thermoregulatory circuitry via two basic pathways, namely the humoral and neural pathways.

The humoral pathway

- In this pathway, fever signals are carried by components of microbial products named pathogen associated molecular patterns (PAMPS) or by pyrogenic cytokines.

- The febrile response is characterised by an early rapid phase and a delayed late phase
- The second humoral pathway is directed by circulating pyrogenic cytokines. They transmit fever signals to the thermoregulatory circuitry by both indirect and direct pathways.

The neural pathway

Peripheral fever signals can communicate with the CNS through peripheral nerves such as cutaneous sensory nerves and the vagus nerve. The activation of the neural pathway is believed to be another mechanism by which fever is rapidly initiated.

Mechanisms underlying fever patterns

- The mechanisms underlying the specificity of the fever patterns to certain diseases are not completely understood. For some infectious diseases it may be related to the life cycle of causative agent. For instance, in malaria, parasites are released into the circulation after 48-72 h of erythrocytic cycle of plasmodium falciparum/ovale/vivax.
- Released parasites activate pyrogenic cytokines which then lead to cycles of fever every 48-72 h (tertian fever).
- However, plasmodium falciparum in contrast to other species may infect multiple red cells in a non-selective manner with each having independent erythrocytic parasite life cycles.
- Consequently, fever due to this parasite is often quotidian (daily fevers spikes).
- Plasmodium vivax/ovale and P. malariae infects young and senescent erythrocytes that rupture to release merozoites (pyrogens) 72- and 96-h respectively. These events partly account for the cyclical nature of fever in these malarial fevers.
- A down regulation of cytokine release after repeated exposure to pyrogens such as LPS may lead to fever remittance or intermittence
- Recurrent fevers may be due to partial treatment of deep seated infections like abscesses or due to recurrent exposure to new antigens (e.g. allergens in hypersensitivity pnuemonitis).
- Such drug (allergens) may manifest with eosinophilia in patients with drug fever. In cyclic neutropenic fevers, febrile episodes correspond to periods of neutropenia and are due to repeated bacterial infections.
- In cancers and pulmonary embolism, fever recurrence has been attributed, partly, to tissue necrosis as phagocytosis of necrotic tissue leads to intermittent release of pyrogenic cytokines.
- Recurrent fevers may be related to the pathogenesis of the disease as exemplified in relapsing fevers due to spirochetes where episodic spirochetemia lead to episodes of fever accompanied by fever free periods which coincides with disappearance of spirochetes from the circulation.
- Night sweats are common in healthy adults but they become clinically significant if associated with fever and drenching.
- They may be attributed to pyrogenic properties of certain diseases which result in early morning fever spikes accompanied hours later by fever remission presenting as night sweats.

Significance of fever patterns

- In clinical practice, many typical fever patterns described above are rarely observed.
- This is as a result of several confounding factors such as ingestion of antipyretics, partial treatment infectious disease-related fevers with antimicrobials and failure to mount fever in the face active infection (for instance the elderly, immunocompromised and severely malnourished individuals may not mount fever).
- Furthermore, the occurrence of concurrent infections by multiple agents in an individual

may alter typical fever patterns associated with specific infectious diseases. For these reasons, the utility of fever patterns in clinical diagnosis is often limited.

- However, when observed, typical fever patterns may provide some useful diagnostic clues for infectious diseases without localising signs such as malaria and typhoid. They may also prove useful in eliminating unlikely diagnosis while directing possible diagnostic investigations to be undertaken for confirmation. In poor resource settings, analysis of fever patterns may provide diagnostic clues for difficult to diagnose infectious diseases especially when appropriate diagnostic investigations are unavailable or unhelpful.
- In the evaluation of pyrexia of unknown origin (PUO) for instance, the presence of low grade intermittent fever and night sweats without localising sign is a useful clue to investigate for extrapulmonary tuberculosis or to initiate empirical anti-tuberculosis therapy in tuberculosis endemic regions.
- The nature of fever defervescence may also provide some diagnostic clues. For instance, in differentiating malaria from typhoid fever, effective anti-malarial therapy leads to fever defervescence by crisis (within hours) whereas in typhoid fevers resolution occurs by lysis (gradually over days) following effective antibiotics.
- However, failure of fever to resolve by crisis does not exclude pre-existing malaria as malaria and typhoid fever may occur concurrently, especially in the tropics.
- It has been suggested that fevers due to cancers can be distinguished from those due infectious diseases using the Naprosyn test.
- When Naprosyn (Naproxen 375 mg twice daily), or other NSAIDS, is given for 3 days, fevers due to cancers display a rapid and sustained decline while little or no change is observed in fevers due to infectious diseases.
- However, Naprosyn test is unhelpful in differentiating neoplastic from noninfectious disorders such as connective tissue diseases.
- Similarly, elevated serum procalcitonin level has been used to differentiate infectious from non-infectious causes of fever with variable results.

Diseases called 'fever'

- Based on its fundamental role in explaining the manifestations of diseases as well as its prominence as a feature of many diseases of man and animals, fever is often included in the appellation of many diseases.
- The naming of diseases called fever are usually based on specific epidemiological and clinical characteristics of the disease including: the way the disease is acquired (e.g. Hay fever is a febrile illness due to exposure to 'hay' in predisposed individuals); the transmitting vector/agent (e.g. cat scratch fever is an infectious disease due to a bite or 'scratch from a cat'); the prominent clinical manifestation (e.g. yellow fever is a viral illness so named because jaundice or 'yellow eyes' is a prominent manifestation in severe cases); the geographical location where the disease was first discovered (e.g. Lassa fever is a haemorrhagic viral infection so named because it was first described in 1969 in a village named Lassa, in Borno state, Nigeria; where it is highly prevalent (e.g. Familial Mediterranean fever is a hereditary febrile disease common among people in the Mediterranean region); where a major outbreak occurred (e.g. Pontiac fever, a non-pneumonic variant of Legionella infection is named after an outbreak in Pontiac, Michigan, in 1968 .

Measles

Before immunisation campaigns, measles occurred in almost 100% of children world wide. The WHO has set the objective of eradicating measles globally by 2010, using the live attenuated vaccine. However, vaccination of only 70-80% of the population, as is currently the case in the UK, for examples, is insufficient to prevent outbreaks in older children and adults, who are more susceptible to complications. Natural illness produces lifelong immunity.

Clinical features

Incubation period-6-19 days

It spreads by respiratory droplets.

Prodromal symptoms

1. Upper respiratory symptoms
2. Conjunctivitis
3. Presence of Koplik's spots on the internal buccal mucosa.

General signs & symptoms

1. The small white spots surrounded by erythema are pathognomonic of measles.
2. As natural antibody develops the maculopapular rash appears, spreading from the face to the extremities.
3. General Lymphadenopathy
4. Diarrhoea

Complications

1. Otitis media
2. Bacterial pneumonia
3. Clinical encephalitis in 0.1% of children
4. Subacute sclerosing panencephalitis (SSPE)

Investigations & Diagnosis

1. Diagnosis is by clinical features
2. Detection of antibody-Serum IgM, Sero Conversion or Salivary IgM.

Note :

1. Disease is more severe and prolonged in adults and complications include pneumonitis, hepatitis and encephalitis.
2. Measles is a serious disease in the malnourished, vitamin deficient or immunocompromised, in whom the typical rash may be missing and persisting infection with a giant cell pneumonitis or encephalitis may occur.
3. Death usually results from bacterial superinfection such as pneumonia, diarrhoea or noma/cancrum oris.

Chickenpox (varicella)

1. Varicella zoster virus (VZV) is a dermotropic and neurotropic virus that produces primary infection, usually in childhood, which may reactivate in later life.
2. VZV is spread by aerosol and direct contact.
3. It is highly infectious to non-immune individuals.
4. Disease in children is usually well tolerated.
5. Manifestations are more severe in adults, pregnant women and the immunocompromised.

Clinical features

1. The incubation period is 11-20 days
2. Vesicular eruption begins, often on mucosal surfaces first, followed by rapid dissemination in a centripetal distribution (most dense on trunk and sparse on limbs).
3. New lesions occur every 2-4 days and each crop is associated with fever.
4. The rash progresses from small pink macules to vesicles and pustules within 24 hours.
5. Infectivity lasts from up to 4 days (but usually 48 hours) before the lesions appear until the last vesicles crust over.

Complications

6. Due to intense itching, secondary bacterial infection from scratching is the most common complication of primary chickenpox.
7. Self-limiting cerebellar ataxia and encephalitis are rare complications.

Risk Factors

8. Adults, pregnant women and the immunocompromised are at increased risk of visceral involvement, which presents as pneumonitis, hepatitis or encephalitis.
9. Pheumonitis can be fatal and is more likely to occur in smokers.
10. Maternal infection in early pregnancy carries a 3% risk of neonatal damage with

developmental abnormalities of eyes, CNS and limbs.

11. Chickenpox within 5 days of delivery leads to severe neonatal varicella with visceral involvement and haemorrhage.

Diagnosis

1. Diagnosis is primarily clinical, by recognition of the rash.
2. If necessary, this can be confirmed by detection of antigen (direct immunofluo-rescence), DNA (PCR) or by viral culture of aspirated vesicular fluid.
3. Serology is used to identify seronegative individuals at risk of infection.

Herpes Simplex Virus Infections

Etiologic Agent

- Herpes simplex viruses (HSV-1, HSV-2; *Herpesvirus hominis*) produce a variety of infections involving mucocutaneous surfaces, the central nervous system (CNS), and—on occasion—visceral organs.
- Prompt recognition and treatment reduce the morbidity and mortality rates associated with HSV infections.

Pathogenesis

Exposure to HSV at mucosal surfaces or abraded skin sites

↓

Permits entry of the virus into cells of the epidermis and dermis

↓

Initiation of viral replication starts

↓

During the initial phase of infection, viral replication occurs in ganglia and contiguous neural tissue.

↓

Virus then spreads to other mucocutaneous surfaces through centrifugal migration of infectious virions via peripheral sensory nerves.

↓

This mode of spread helps explain the large surface area involved, the high frequency of new lesions distant from the initial crop of vesicles that is characteristic in patients with primary genital or oral-labial HSV infection,

↓

And the ability to recover virus from neural tissue distant from neurons innervating the inoculation site.

↓

Contiguous spread of locally inoculated virus also may take place and allow further mucosal extension of disease.

Clinical Spectrum

1. HSV has been isolated from nearly all visceral and mucocutaneous sites.
2. The incubation period ranges from 1 to 26 days (median, 6–8 days).
3. Both viral subtypes can cause genital and oral-facial infections, and the infections caused by the two subtypes are clinically indistinguishable.
4. Genital HSV-2 infection is twice as likely to reactivate and recurs 8–10 times more frequently than genital HSV-1 infection.
5. Oral-labial HSV-1 infection recurs more frequently than oral-labial HSV-2 infection.

Oral-Facial Infections

- Gingivostomatitis and pharyngitis are the most common clinical manifestations of first-episode HSV-1 infection, while recurrent herpes labialis is the most common clinical manifestation of reactivation HSV-1 infection.
- HSV-1 and varicella-zoster virus (VZV) have been implicated in the etiology of Bell's palsy (flaccid paralysis of the mandibular portion of the facial nerve).

Genital Infections

1. First-episode primary genital herpes is characterized by fever, headache, malaise, and myalgias.
2. Pain, itching, dysuria, vaginal and urethral discharge, and tender inguinal

lymphadenopathy are the predominant local symptoms.

Herpetic Whitlow

Herpetic whitlow—HSV infection of the finger—may occur as a complication of primary oral or genital herpes by inoculation of virus through a break in the epidermal surface or by direct introduction of virus into the hand through occupational or some other type of exposure.

Herpes Gladiatorum

HSV may infect almost any area of skin. Mucocutaneous HSV infections of the thorax, ears, face, and hands have been described among wrestlers.

Eye Infections

HSV infection of the eye is the most common cause of corneal blindness in the United States. HSV keratitis presents as an acute onset of pain, blurred vision, chemosis, conjunctivitis, and characteristic dendritic lesions of the cornea.

Central and Peripheral Nervous System Infections

HSV accounts for 10–20% of all cases of sporadic viral encephalitis.

Visceral Infections

HSV infection of visceral organs usually results from viremia, and multiple-organ involvement is common. Occasionally, however, the clinical manifestations of HSV infection involve only the esophagus, lung, or liver.

Neonatal HSV Infections

Neonates (infants younger than 6 weeks) have the highest frequency of visceral and/or CNS infection.

HSV in Pregnancy

The risk of mother-to-child transmission of HSV in the perinatal period is highest when the infection is acquired near the time of labor—that is, in previously HSV-seronegative women.

Diagnosis-Investigations

1. Both clinical and laboratory criteria are useful for diagnosing HSV infections.
2. Staining of scrapings from the base of the lesions with Wright's, Giemsa's (Tzanck preparation), or Papanicolaou's stain to detect giant cells or intranuclear inclusions of *Herpes virus* infection
3. HSV infection is best confirmed in the laboratory by detection of virus, viral antigen, or viral DNA in scrapings from lesions.
4. HSV DNA detection by PCR is the most sensitive laboratory technique for detecting mucosal or visceral HSV infections
5. HSV causes a discernible cytopathic effect in a variety of cell culture systems, and this effect can be identified within 48–96 h after inoculation.
6. Spin-amplified culture with subsequent staining for HSV antigen has shortened the time needed to identify HSV to <24 h.

Laboratory confirmation permits subtyping of the virus; information on subtype may be useful epidemiologically and may help to predict the frequency of reactivation after first-episode oral-labial or genital HSV infection.

Herpes zoster

Herpes zoster is usually a disease of older people. It is commonly known as shingles and also known as zona, is a viral disease characterized by a painful skin rash with blisters in a limited area on one side of the body, often in a stripe. Throughout the world, the incidence rate of herpes zoster every year ranges from 1.2 to 3.4 cases per 1,000 healthy individuals, increasing to 3.9–11.8 per year per 1,000 individuals among those older than 65 years. It is a self-limiting disorder but unlike chicken pox the rash is very painful and even after it disappears, the pain may persist specially in the elderly due to post herpetic neuralgia. .

Signs and Symptoms

- Headache
- fever
- malaise
- burning pain

- itching
- hyperesthesia or paresthesia

Herpes zoster may have additional symptoms, depending on the dermatome involved. *Herpes zoster ophthalmicus* involves the orbit of the eye and occurs in approximately 10 to 25% of cases. It is caused by the virus reactivating in the ophthalmic division of the trigeminal nerve. In a few patients, symptoms may include conjunctivitis, keratitis, uveitis, and optic nerve palsies that can sometimes cause chronic ocular inflammation, loss of vision, and debilitating pain. *Herpes zoster oticus,* also known as Ramsay Hunt syndrome type II, involves the ear. It is thought to result from the virus spreading from the facial nerve to the vestibulo-cochlear nerve. Symptoms include hearing loss and vertigo (rotational dizziness).

Coxsackieviruses

- Coxsackieviruses belong to the family Picornaviridae and the genus Enterovirus, which also includes poliovirus and echovirus.
- Enteroviruses are among the most common and important human pathogens with 9 different species classified (Enterovirus A-H and I).

Pathophysiology

- Coxsackieviruses primarily are transmitted via the fecal-oral route and respiratory aerosols, although transmission via fomites also is possible.
- Viral replication initially occurs in the upper respiratory tract, particularly the tonsils, and the distal small bowel.
- Viremia with systemic dissemination then occurs and involves the reticuloendothelial system, which leads to further replication in other anatomic locations and organs and ultimately to development of symptoms.
- Central nervous system (CNS) invasion is proposed to occur from viral migration along peripheral and central nerves into the CNS.
- Coxsackieviruses have been identified in the respiratory tract up to 3 weeks after initial infection and in feces up to 8 weeks after initial infection. Innate, humoral and cell-mediated immunity all play a role in the body's response to enteroviral infection. However, the infection frequently is eradicated before antibody production occurs

Clinical Presentation

- More than 90% of coxsackieviruses infections are asymptomatic or cause nonspecific febrile illnesses.
- In neonates, they are the most common cause of febrile illnesses during the summer and fall months.
- As measured year round, thirteen percent of newborns with fever in the first month of life were noted to have an enteroviral infection.

In addition to nonspecific febrile illnesses, various well-described syndromes also have been associated with coxsackievirus infections.

- Aseptic meningitis
- Encephalitis
- Other neurologic syndromes
- Rarely, coxsackieviruses have been implicated in other sporadic neurologic syndromes such as acute flaccid myelitis (AFM) that closely mimics poliovirus infection.
- Myopericarditis
- Diabetes
- Exanthems and enanthems
- Two of the most distinctive rash syndromes caused by coxsackieviruses are hand, foot, and mouth disease (HFMD) and herpangina.
- Epidemic pleurodynia
- Acute hemorrhagic conjunctivitis
- Sepsis

Ancillary investigations

- Enteroviruses can be excreted in human feces for up to 3 months after infection.
- However, a clinically identifiable syndrome correlates with the acute phase of infection, during which time virus can be found in the throat, blood, and various organs.

- There are no confirmatory laboratory tests, procedures, or imaging that are used in routine clinical practice for HFMD or herpangina.
- Diagnosis for these conditions mainly is based on clinical presentation and assessment.

Rubella (German measles)

- Rubella is an endemic exanthem in countries without universal vaccination.
- In non-immunised communities 80-85% of young adults have evidence of past infection.

Aetiology

- Rubella and congenital rubella syndrome are caused by rubella virus.
- Only one antigenic type of rubella virus is available
- The humans are the only natural hosts

Clinical features

1. Rubella is spread by respiratory droplet, with infectivity from up to 10 days before to 2 weeks after the onset of the rash.
2. The incubation period is 15-20 days.
3. In childhood, most cases are subclinical, although clinical features may include fever, maculopapular rash spreading from the face, and lymphadenopathy.
4. Complications are rare but include thrombocytopenia and hepatitis.
5. Encephalitis and haemorrhage are occasionally reported.
6. In adults, arthritis involving hands or knees is relatively common, especially in women.

Rubella infection: risk of congenital malformation

Stage of gestation	*Likelihood of malformations*
1-2 months	65-85% chance of illness, multiple defects/ spontaneous abortion
3 months	30-35% chance of illness, usually a single congenital defect (most frequently deafness, cataract, glaucoma, mental retardation, congenital heart disease, especially pulmonary stenosis, patent ducts arteriosus)
4 months	10% risk of congenital defects, most commonly deafness
> 20 weeks	Occasional deafness

Diagnosis

1. Laboratory confirmation of rubella is required if there has been contact with a pregnant woman.
2. This is achieved either by detection of rubella IgM in serum or by IgG seroconversion.
3. In the exposed pregnant woman, absence of rubella-specific IgG confirms the potential for congenital infection.

Hand, foot, and mouth disease (HFMD)

- HFMD often affects children and easily spreads to close contacts.
- Patients present with a sore throat and painful lesions in the mouth.
- Vesicles that coalesce, form bullae, and then ulcerate, occur on the buccal mucosa and tongue. Seventy-five percent of patients have simultaneous peripheral cutaneous lesions.
- HFMD also can present more atypically, including as eczema coxsackium, with erosions or bullous lesions.
- Biopsy reveals intracytoplasmic viral particles. Coxsackieviruses A16, A6, and A10 are among the most common types implicated in HFMD.
- Numerous cases of a more severe HFMD caused by coxsackievirus A6 were reported between 2004 and 2011 in several Asian and European countries. Additionally, between 2011 and 2012 in the United States, among cases of severe HFMD that were reported, 74% had a positive PCR test for coxsackievirus A6; about 25% of reported cases occured in adults.

Filariasis

Filariasis is a group of disorders caused by infection with the thread-like nematodes. Lymphatic filariasis is caused by *Wuchereria bancrofti* and *Brugia malayi*. Light infections may be completely asymptomatic.

Symptoms

The clinical manifestations are:

- o Low grade fever which may be accompanied by chills and sweats
- o lymphangitis, involving the legs more frequently than the arms
- o lymphadenitis, involving inguinal, femoral and epitrochlear nodes
- o dilatation of lymphatic which may rupture
- o chyluria
- o lymphedema of one or more extremities or other parts
- o episodes of secondary bacterial infection
- o tropical eosinophilia.

Diagnosis: Microfilariae may be detected in peripheral smear (night sample), Serological tests are not reliable.

Malaria

1. Malaria is a protozoan disease transmitted by the bite of infected *Anopheles* mosquitoes
2. Malaria in humans is caused by plasmodium falcifarum, P.Vivax, P. Ovale, P. malariae and the predominantly simian parasite, P Knowles.
3. It is transmitted by the bite of female anopheline mosquitoes and occurs throughout the tropics and subtropics at altitudes below 1500 metres.
4. Travellers are susceptible to malaria.
5. A few people living near airports in Europe have acquired malaria from accidentally imported mosquitoes.

Pathogenesis

Life cycle of the malarial parasite

The female *Anopheles* mosquito becomes infected when it feeds on human blood containing gametocytes, the sexual forms of the malarial parasite

↓

Development in the mosquito takes from 7 to 20 days

↓

Results in sporozoites accumulating in the salivary glands

↓

Inoculate into the human blood stream

↓

Sporozoites disappear from human blood within half an hour

↓

Enter the liver.

↓

After some days merozoites leave the liver and invade red blood cells

↓

Where further asexual cycles of multiplication take place,

↓

Resulting into the production of schizonts

↓

Rupture of the schizont releases more merozoites into the blood

↓

Causes Malaria fever
(The periodicity of which depends on the species of parasite)

High yield information

P. Vivax, P. Ovale may persist in liver cells as dormant forms, hypnozoites, capable of developing into merozoites months or years later. Thus the first attack of clinical malaria may occur long after, the patient has left the endemic area, and the disease may relapse after treatment if drugs that kill only the erythrocytic stage of the parasite are given.

Plasmodium falcifarum and P. malariae have no persistent exoerythrocytic phase but recrudescence of fever may result from multiplication of parasites in red cells which have not been eliminated by treatment and immune processes.

Pathology

Red cells infected with malaria are prone to haemolysis

↓

It is most severe with Plasmodium falcifarum because it invades red cells of all ages but especially young cells

↓

P.Vivax, , P.ovale invade reticulocytes

↓

P. Malariae normoblasts

↓

Anaemia may be profound and is worsened by dyserythropoiesis, splenomegaly and depletion of folate stores

↓

P. falcifarum malaria, red cells containing trophozoites adhere to vascular endothelium in post-capillary venules in brain, kidney, liver, lungs and gut.

↓

The vessels become congested, resulting in widespread organ damage which is exacerbated by rupture of schizonts, liberating toxic and antigenic substances

↓

Hence P. falcifarum infection is dangerous and fatal with increased mortality and morbidity

Clinical features

P. falciparum infection

1. This is the most dangerous of the malarias and patients are either 'killed or cured'
2. The onset is often insidious, with malaise; headache and vomiting associated with Cough and mild diarrhoea are also common.
3. The fever has no particular pattern.
4. Jaundice is common due to haemolysis and hepatic dysfunction.
5. The liver and spleen enlarge and may become tender.
6. Anaemia develops rapidly, as does thrombocytopenia
7. May rapidly develop dangerous complications
8. Cerebral malaria is manifested by confusion, seizures or coma, usually without localising signs.
9. Children die rapidly without any special symptoms other than fever. F
10. Immunity is impaired in pregnancy and the parasite can preferentially bind to a placental protein known as chondroitin sulphate A.
11. Abortion and intrauterine growth retardation from parasitisation of the maternal side of the placenta are frequent.
12. Previous splenectomy increases the risk of severe malaria.

P. vivax and P ovale infection

1. In many cases the illness starts with several days of continued fever before the development of classical bouts of fever on alternate days.
2. Fever starts with a rigor. The patient feels cold and the temperature rises to about 40^{0}C.
3. After half an hour to an hour the hot or flush Phase begins.
4. It lasts several hours and gives way to profuse perspiration and a gradual fall in temperature.
5. The cycle is repeated 48 hours later.
6. Gradually the spleen and liver enlarge and may become tender.
7. Anaemia develops slowly.
8. Relapses are frequent in the first 2 years after leaving the malarious area and infection may be acquired from blood transfusion.

P. malariae infection

1. This is usually associated with mild symptoms and bouts of fever every third day
2. Parasitemia may persist for many years with the occasional recrudescence of Fever or without producing any symptoms.
3. Chronic *P malariae* infection causes glomerulonephritis and longterm nephrotic syndrome in children.

Investigations

Peripheral blood examination

- Giemsa-stained thick and thin blood films should be examined whenever malaria is suspected.

- In the thick film erythrocytes are lysed, releasing all blood stages of the Parasite.
- A thin film is essential to confirm the diagnosis, to identify the species of parasite and in P. falciparum infections.

Immunochromatographic tests

- Immunochromatographic tests for malaria antigens, such as Optimal (which detects the Plasmodium lactate dehydrogenase of several species)
- Parasight F (which detects the P. Falciparum)

PfHRP2 dipstick or card test

- A drop of blood is placed on the stick or card, which is then immersed in washing solutions.
- Monoclonal antibody captures the parasite antigen and reads out as a colored band.

Disadvantages

- Detects only *Plasmodium falciparum*; remains positive for weeks after infection; does not quantitate *P. falciparum* parasitemia

Plasmodium LDH dipstick or card test

- A drop of blood is placed on the stick or card, which is then immersed in washing solutions.
- Monoclonal antibodies capture the parasite antigens and read out as colored bands. One band is genus specific (all malarias), and the other is specific for *P. falciparum.*

Disadvantages

Slightly more difficult preparation than PfHRP2 tests; may miss low-level parasitemia with *P. vivax*, *P. ovale*, and *P. malariae* and does not speciate these organisms; does not quantitate *P. falciparum* parasitemia

Microtube concentration methods with acridine orange staining

- Blood is collected in a specialized tube containing acridine orange, anticoagulant, and a float.
- After centrifugation, which concentrates the parasitized cells around the float, fluorescence microscopy is performed.

Full blood count and general *blood* picture

- Normochromic, normocytic anemia is usual.
- The leukocyte count is generally normal, although it may be raised in very severe infections.
- There is slight monocytosis, lymphopenia, and eosinopenia, with reactive lymphocytosis and eosinophilia in the weeks after the acute infection.
- The platelet count is usually reduced to -10^5/ μL.

ESR and CRP

- The erythrocyte sedimentation rate, plasma viscosity, and levels of C-reactive protein and other acute-phase proteins are high.

Prothrombin and partial thromboplastin times

- Severe infections may be accompanied by prolonged prothrombin and partial thromboplastin times and by more severe thrombocytopenia.
- Levels of antithrombin III are reduced even in mild infection.

Electrolytes, blood urea nitrogen (BUN), and creatinine

- In uncomplicated malaria, plasma concentrations of electrolytes, blood urea nitrogen (BUN), and creatinine are usually normal.
- metabolic acidosis, with low plasma concentrations of glucose, sodium, bicarbonate, calcium, phosphate, and albumin together with elevations in lactate, BUN, creatinine, urate, muscle and liver enzymes, and conjugated and unconjugated bilirubin may be observed in severe malaria

Hypergammaglobulinemia

- Hypergammaglobulinemia is usual in immune and semi-immune subjects.

Urinalysis

- Urinalysis generally gives normal results.

Cerebrospinal fluid (CSF)

In adults and children with cerebral malaria, the mean opening pressure at lumbar puncture is 160 mm of cerebrospinal fluid (CSF); usually the CSF is normal or has a slightly elevated total protein level [<1.0 g/L (<100 mg/dL)] and cell count (<20/ L).

Influenza

Definition

- Influenza is an acute respiratory illness caused by infection with influenza viruses.
- The illness affects the upper and/or lower respiratory tract and is often accompanied by systemic signs and symptoms such as fever, headache, myalgia, and weakness.
- Outbreaks of illness of variable extent and severity occur nearly every year.
- Such outbreaks result in significant morbidity rates in the general population and in increased mortality rates among certain high-risk patients, mainly as a result of pulmonary complications.

Aetiology

1. Influenza is an acute systemic viral infection that primarily affects the respiratory tract and it carries a significant mortality.
2. It is caused by influenza A virus or influenza B virus (milder form).
3. Infection is seasonal, and variation in the haemagglutinin (H) and neuraminidase (N) glycoproteins on the surface of the virus leads to disease of variable intensity each year.
4. Nomenclature of influenza strains is based on these glycoproteins, e.g. H1 N1, H3N2 etc.
5. Genetic shift results in the circulation of a new influenza strain within a community to which few people are immune, potentially initiating an influenza epidemic or pandemic in which there is a high attack rate and there may be increased disease severity.

Clinical features

1. Incubation period -1-3 days
2. Uncomplicated disease leads to fever, malaise and cough.
3. Viral pneumonia may occur, although pulmonary complications are most often due to superinfection with *Strep.pneumoniae, Staph. aureus* or other bacteria.
4. Rare extra pulmonary manifestations include myositis, myocarditis, pericarditis and neurological complications (Reye's syndrome in children, encephalitis or transverse myelitis).

Diagnosis / Investigations

Acute infection is diagnosed by viral Culture, or by antigen or RNA detection (reverse transcription RT)-PCR) in a nasopharyngeal sample. The disease may also be diagnosed retrospectively on the basis of seroconversion.

Avian influenza

1. Avian influenza is caused by influenza A viruses with alternative haemagglutinin antigens, including the H5N1 strain.
2. These viruses have an increased ability to bind to lower respiratory tract epithelium, causing more severe disease with increased incidence of viral pneumonia and respiratory failure.
3. The majority of cases have occurred in individuals with a history of exposure to poultry, predominantly in South-east Asia.
4. Recent 'flu' seasons, cases have spread further west and infection has been identified in Europe in migrating birds and imported poultry.
5. Existing strains have been associated with infrequent person-to-person transmission but there is a concern that adaptation of an avian strain to allow effective person-to-person transmission is likely to lead to a global pandemic of life-threatening influenza.

Swine influenza

1. Occasional cases of influenza are transmitted from pigs to humans.
2. An outbreak of swine flu began in 2009,

initially in Mexico and then spreading around the world.

3. The causative strain was shown to be an H1N1 strain which showed significant genetic variation from human strains of HI N1.
4. Clinical features of infections with this strain are typical of influenza A infection, although some cases have more pronounced enteric features.

Infectious mononucleosis (IM) and Epstein-Barr virus (EBV)

- Epstein-Barr virus (EBV) is the cause of heterophile-positive infectious mononucleosis (IM), which is characterized by fever, sore throat, lymphadenopathy, and atypical lymphocytosis.
- EBV is also associated with several human tumors, including nasopharyngeal carcinoma, Burkitt's lymphoma, Hodgkin's disease, and (in patients with immunodeficiencies) B cell lymphoma. The virus is a member of the family Herpesviridae. The two types of EBV that are widely prevalent in nature are not distinguishable by conventional serologic tests.
- About 50% of infections result in typical IM.
- The virus is usually acquired from asymptomatic excretes via saliva, either by droplet infection or environmental contamination in childhood, or by kissing among adolescents and adults.
- EBV is not highly contagious..

Clinical features

1. IM has a prolonged and undetermined incubation period
2. Fever, headache and malaise.
3. This is followed by severe pharyngitis, which may include tonsillar exudates, and non-tender interior and posterior cervical lymphedenopathy.
4. Palatal petechiae, periorbital odema, splenomegaly, inguinal or axillary lymphadenopathy, and macular, petechial or erythema multiform rashes may occur.
5. In most cases fever resolves over 2 weeks, and fatigue and other abnormalities settle over a further few weeks.
6. Death is rare but can occur due to respiratory obstruction, haemorrhage from splenic rupture or thrombocytopenia, or encephalitis.

Diagnosis

- The white blood cell count is usually elevated and peaks at 10,000–20,000/L during the second or third week of illness.
- Lymphocytosis is usually demonstrable, with >10% atypical lymphocytes.
- The latter cells are enlarged lymphocytes that have abundant cytoplasm, vacuoles, and indentations of the cell membrane
- CD8+ cells predominate among the atypical lymphocytes.
- Low-grade neutropenia and thrombocytopenia are common during the first month of illness.

Liver function test

- Liver function is abnormal in >90% of cases.
- Serum levels of aminotransferases and alkaline phosphatase are usually mildly elevated.
- The serum concentration of bilirubin is elevated in 40% of cases.

Dengue

Etiology

1. The principal vector is the mosquito Aedes aegypti, which breeds in standing water, collections of water in containers, water-based air coolers and tyre dumps are a good environment for the vector in large cities.
2. Aedes albopictus is a vector in some South-east Asian countries.
3. There are four serotypes of dengue virus, all producing a similar clinical syndrome;
4. Homonymic immunity after infection with one of the serotypes is life-long, but heterotypic immunity against the other serotypes lasts only a few months after infection.

Clinical features of dengue fever

Prodrome : 2 days of malaise and headache

Acute onset : Fever, backache, arthralgias, headache, generalised pains ('break-bone fever'), pain on eye movement, lacrimation, anorexia, nausea, vomiting, relative bradycardia, prostration, depression, lymphadenopathy, scleral injection

Fever : Continuous or 'saddle-back', with break on 4th or 5th day and then recrudescence; usually lasts 7-8 days

Rash : Initial flushing faint macular rash in first 1-2 days. Maculopapular, scarlet morbilliform rash from days 3-5 on trunk, spreading centrifugally and sparing palms and soles, onset often with fever defervescence. May desquamate on resolution or give rise to petechiae on extensor surfaces.

Convalescence

- Slow

Complications

Minor bleeding from mucosal sites, hepatitis, cerebral hemorrhage or oedema, rhabdomyolysis.

1. The incubation period from being bitten by an infected mosquito is usually 2-7 days.
2. Asymptomatic infections are common but the disease is more severe in infants and the elder.
3. A morbilliform rash, which characteristically blanches under pressure, may occur, often as the fever is settling.
4. A more severe illness, called dengue haemorrhagic fever or dengue shock syndrome, occurs mainly in children in South-east Asia
5. In mild forms, there is thrombocytopenia and haemoconcentration.
6. In the most severe form, after 3-4 days of fever, hypotension and circulatory failure develop with pleural effusions, ascites, hypoalbuminaemia and features of acute respiratory distress syndrome.
7. Minor (petechiae, ecchymoses, epistaxis) or major (gastrointestinal or cerebrovascular) haemorrhagic signs may occur.

Diagnosis

1. Leucopoenia is usual and thrombocytopenia common.
2. The diagnosis is confirmed by either at fourfold rise in the antibody titres, isolation of dengue virus from blood or detection of dengue virus RNA by PCR.
3. Serological tests may detect cross-reacting antibodies against other flavi viruses, including yellow fever vaccine.

Leptospirosis

- Leptospirosis is an infectious disease of humans and animals that is caused by pathogenic spirochetes of the genus Leptospira.
- It is considered the most common zoonosis in the world and is associated with settings of poor sanitation and agricultural occupations involving contact with animals or water.
- Increasingly, adventurous travel and "mud run" sports or races involving fresh water or soil exposure put humans at risk. Affected reservoir species experience chronic colonization of proximal renal tubules and shed leptospires in urine. Humans are not typically considered reservoirs but may chronically shed leptospires and may act as reservoirs in areas of high endemicity and poor waste sanitiation.
- Leptospirosis is endemic in most areas where dengue virus is transmitted and may be mistaken for dengue, which is typically more common. Co-infection occurs in up to 8% of cases. Leptospirosis may also co-infect with dengue and other regionally prevalent pathogens, such as malaria, hantavirus, and scrub typhus.
- Leptospirosis is typically a biphasic pattern of early flu-like, septicemic illness followed by an inflammatory second phase. The latter may be characterized by systemic inflammatory response syndrome (SIRS) or cytokine storm. COVID-19 may follow a similar trajectory and may complicate diagnosis in the subtropics and tropics. These issues may complicate the diagnosis, especially in travelers or migrants presenting to non-endemic areas. It is important to consider leptospirosis when dengue is diagnosed in a severely ill patient, because early antibiotics are beneficial.

Etiology

- Leptospirosis is caused by spiral bacteria that belong to the genus Leptospira, the family Leptospiraceae, and the order Spirochaetales. These spirochetes are finely coiled, thin, motile, obligate, slow-growing aerobes.
- The traditional system divided the genus into 2 species: the pathogenic Leptospira interrogans and the nonpathogenic Leptospira biflexa. These species were divided further into serogroups, serovars, and strains based on shared antigens. L interrogans included more than 250 serovars.
- Nevertheless, any leptospiral serovar can lead to the signs and symptoms seen with this disease. For example, jaundice occurs in 83% of patients with L icterohaemorrhagiae infection and in 30% of patients infected with L pomona. Aseptic meningitis commonly occurs in those infected with L pomona or L canicola.
- Transmission of leptospires to humans typically occurs by invasion across mucosal surfaces or nonintact skin. Infection may occur via direct contact with infected animals or their tissues or urine or through contact with contaminated water and soil.

Clinical presentations

- Onset of clinical illness occurs abruptly, after an incubation period of 2-30 days (typically 5-14 d).
- Expert consensus is that leptospirosis occurs as two recognizable clinical syndromes: anicteric and icteric (the existence of a third syndrome of asymptomatic infection is more controversial).
- Anicteric leptospirosis is a self-limited, mild flulike illness.
- Icteric leptospirosis, also known as Weil disease, is a severe illness characterized by multiorgan involvement or even failure.
- Two distinct phases of illness are observed in the mild form: the septicemic (acute) phase and the immune (delayed) phase. In icteric leptospirosis, the 2 phases of illness are often continuous and indistinguishable. At disease onset, clinically predicting the severity of disease is not possible.
- An acute illness follows infection with any serovar of leptospirosis.

Most of the following signs and symptoms may develop in varying degrees:

- Headache
- Fever (38-40^{0}C)
- Rigors
- Muscle pain (typically localized to the calf and lumbar areas)
- Nausea and vomiting
- Anorexia
- Diarrhea
- Dry cough
- Pharyngitis
- Conjunctival suffusion
- Nonpruritic skin rash

The natural course of leptospirosis falls into 2 distinct phases:

- The acute phase of illness lasts 5-7 days and is followed by a 1-3 day period of improvement in which the temperature curve falls and the patient may become afebrile and relatively asymptomatic. Subsequently, leptospirosis either regresses to a relatively asymptomatic illness or progresses to a more severe illness.
- Recurrence of fever indicates the onset of the second, immune stage. Nonspecific symptoms, such as fever and myalgia, may be less severe than in the first stage and last a few days to a few weeks. Many patients (77%) experience headache that is intense and poorly controlled by analgesics; this often heralds the onset of meningitis.

Investigations

- Leptospires grow slowly in culture, and recovery rates are low.
- Serologic tests are available only in specialized laboratories, and the sensitivity of acute serologic tests is low.

- Laboratory studies are used for two purposes: to confirm the diagnosis and to determine the extent of organ involvement and severity of complications. Laboratory confirmation of leptospirosis can be accomplished through isolation of the pathogen or by serologic testing.
- Isolation of the leptospires from human tissue or body fluids is the criterion standard, but culture is not routinely available.
- Urine is the most reliable body fluid to study because the urine contains leptospires from the onset of clinical symptoms until at least the third week of infection.
- Other body fluids contain the organism, but the window of opportunity to isolate them is shorter.
- Blood and CSF may produce positive PCR or cultures during the first 7-10 days of symptoms.
- Tissues (ie, liver, muscle, kidney, skin, eyes) are also sources of identification of the leptospires but are obviously more complicated to acquire.
- Antileptospire antibodies in these samples are detected using the microscopic agglutination test (MAT).

A complete blood cell count (CBC) is necessary. Findings on general laboratory studies are as follows:

- In patients with mild disease, elevated erythrocyte sedimentation rates and peripheral leukocytosis (3,000-26,000 x 109/L) with a left shift are noted
- Significant anemia due to pulmonary and gastrointestinal hemorrhage can occur
- The platelet count may be diminished as a component of disseminated intravascular coagulation (DIC)
- Levels of blood urea nitrogen and serum creatinine may be profoundly elevated in the anuric or oliguric phase
- Serum creatine kinase levels (MM fraction) are often elevated in patients with muscular involvement.
- Coagulation times may be prolonged in patients with hepatic dysfunction and/or DIC On liver function testing, serum bilirubin levels elevate as part of the obstructive disease due to capillaritis in the liver. Levels of hepatocellular transaminases are elevated less often and less significantly (usually < 200 U/L). Jaundice and bilirubinemia disproportional to hepatocellular damage is common in leptospirosis; alkaline phosphatase levels may be elevated 10-fold.
- On urinalysis, proteinuria may be present. Leukocytes, erythrocytes, hyaline casts, and granular casts may be present in the urinary sediment. Rapid urine antigen test using lateral flow assay is under development.
- Analysis of the CSF is useful only in excluding other causes of bacterial meningitis. When the CNS becomes involved in leptospirosis, polymorphonuclear leukocytes initially predominate and are later replaced by monocytes. CSF protein may be normal or elevated, whereas glucose levels remain normal. CSF pressure is normal, but a lumbar puncture can relieve the headache. Leptospires are routinely isolated from the CSF, but this finding does not change management of the disease.
- Imaging studies are useful in determining the extent and severity of organ involvement. This may include chest radiography or computed tomography to evaluate lung disease and biliary tract ultrasonography in suspected acalculous cholecystitis.
- Electrocardiographic (ECG) abnormalities are common during the leptospiremic phase of Weil syndrome. In severe cases, congestive heart failure and cardiogenic shock may occur.

Chikungunya virus

Aetiology

1. It is an alpha virus that causes fever, rash and arthropathy.
2. It is found principally in Africa and Asia, including India.

3. Humans and non-human primates are the mom reservoir and the main vector is the Aedes aegypti mosquito.
4. Cases occur in epidemics on a background of sporadic cases.

Incubation period

The incubation period is 2-12 days.

Clinical features

1. A period of fever may be followed by an afebrile phase and then recrudescence of fever.
2. Children may develop maculo-papular rash.
3. Adults are susceptible to arthritis, which causes early morning pain and swelling, must often in the small joints.
4. Arthritis can persist for months and may become chronic in HLA-B27-positive individuals.
5. Related alpha virus causing similar syndromes include Sindbis virus (Scandinavia and Africa), O'nyong-nyong virus (Central Africa), Ross River virus (Australia) and Mayaro virus (Caribbean ami South America).
6. A related virus, O'nyong-nyong, caused a major epidemic of arthritis and rash involving at least 2 million people as it moved across eastern and central Africa in the 1960s.
7. After its mysterious emergence, the virus virtually disappeared, leaving only occasional evidence of its persistence in Kenya until a transient resurgence of epidemic activity in 1997

Diagnosis

- It is done by serology but cross-reactivity between alpha viruses occurs.
- The rash is most intense on the trunk and limbs and may desquamate.
- Petechiae are occasionally seen, and epistaxis is not uncommon, but this virus is not a regular cause of the HF syndrome, even in children.

Full blood count

- A few patients develop leukopenia.
- mildly decreased platelet counts

LFT

- Elevated levels of aspartate aminotransferase (AST) have been described

C-reactive protein

- Elevated levels of C-reactive protein have been described

Typhoid fever / Enteric fever

Etiology

Typhoid and paratyphoid fevers, which are transmitted by the faecal-oral route, are important causes of fever in India, sub-Saharan Africa and Latin America. Enteric fevers are caused by infection with Salmonella typhi and S. Paratyphi A and B.

Pathogenesis

After a few days of bacteraemia

↓

The bacilli localise—mainly in the lymphoid tissue of the small intestine

↓

Resulting in typical lesions in the Peyer's patches and follicles leading to swelling

↓

Ulceration in the Peyer's patches and follicles of the small intestine

(Haemorrhage or a perforation of the ulcerated patches my occur at the end of the 2nd week or during the 3rd week of the illness as a complication)

↓

Usually heal

(After clinical recovery about 5% of patients become chronic carriers; the bacilli may live in the gallbladder for months or years and pass intermittently in the stool and less commonly in the urine)

Clinical features Typhoid fever

Clinical features of typhoid fever :

1st week

- Fever
- Relative bradycardia

- Headache
- Myalgia
- Constipation
- Diarrhoea and vomiting in children

End of 1st week

- Rose spots on turn
- Splenomegaly
- Cough
- Abdominal distension
- Diarrhoea

End of 2nd week

- Delirium, complications, then coma and death (if untreated)

Complications of typhoid fever

Bowel

- Perforation
- Hemorrhage

Septicaemic foci

- Bone and joint infection
- Cholecystitis
- Meningitis

Toxic phenomena

- Myocarditis
- Nephritis

Persistent gallbladder carriage

The incubation period is about 10-14 days and the onset may be insidious. The temperature rises in a stepladder fashion for 4 or 5 day,, with malaise, increasing headache, drowsiness and aching in the limbs. Constipation may be present, although in children diarrhoea and vomiting may be prominent early in the illness. The pulse is often slower than would be expected from the licit, of the temperature, i.e. a relative bradycardia.

At the end of the first week, a rash may appear- on the upper abdomen and on the: back as sparse, slightly raised, rose-red spots, which fade on pressure. It is usually visible only on white skin. Cough and epistaxis occur. Around the 7th-10th day the spleen becomes palpable. Constipation is then succeeded by diarrhoea and abdominal distension with tenderness. Bronchitis and delirium may develop. If untreated, by the end of the 2nd week the patient may be profoundly ill.

Paratyphoid fever

The course tends to be shorter and milder than that of typhoid fever and the onset is often more abrupt with acute enteritis. The rash may be more abundant intestinal complications less frequent.

Complications

1. Haemorrhage or a perforation of the ulcerated patches my occur at the end of the 2nd week or during the 3rd week of the illness
2. Additional complications may involve almost any viscous or system because of the septicaemia present during the 1st week. Bone and joint infection is common in children with sickle-cell disease.

Diagnosis/investigations

Culture (blood, bone marrow, intestinal secretions and stool)

- Other than a positive culture, no specific laboratory test is diagnostic for enteric fever.
- The definitive diagnosis of enteric fever requires the isolation of *S.* typhi or *S.* paratyphi from blood, bone marrow, other sterile sites, rose spots, stool, or intestinal secretions.
- The sensitivity of blood culture is only 40–80%.
- Bone marrow culture is 55–90% sensitive.
- Culture of intestinal secretions (best obtained by a noninvasive duodenal string test) can be positive despite a negative bone marrow culture.
- If blood, bone marrow, and intestinal secretions are all cultured, the yield is >90%.
- Stool cultures, while negative in 60–70% of cases during the first week, can become positive during the third week of infection in untreated patients.

Full Blood count

- In 15–25% of cases, leukopenia and neutropenia are detectable.
- Leukocytosis is more common among children, during the first 10 days of illness, and in cases complicated by intestinal perforation or secondary infection.

LFT and muscle enzyme levels

- Moderately elevated liver function tests and muscle enzyme levels.

Serologic tests

- The classic Widal tests for "febrile agglutinins", are available.
- None of these tests is sufficiently sensitive or specific to replace culture-based methods for the diagnosis of enteric fever in developed countries.

PCR and DNA probe assays

- PCR and DNA probe assays to detect *S. typhi* in blood have been identified but have not yet been developed for clinical use.

MCQs

1. Fever having twice daily spikes is
   a. T.B
   b. Leishmaniasis
   c. Familial meditteranean fever
   d. Amoebiasis
2. Saddle back fever is seen in
   a. Salmonella infection
   b. T.B
   c. CMV infectiion
   d. Dengue fever
3. Kissing's disease (Glandular fever) is caused by
   a. Streptococcus
   b. Staphylococcus
   c. Salmonella
   d. Ebstein Barr Virus
4. Which one is correct in relation to malaria?
   a. The swollen infected liver cell eventually bursts, discharging motile merozoites into the bloodstream to produce malaria
   b. The swollen infected liver cell eventually bursts, discharging motile sporozoites into the bloodstream to produce malaria
   c. A single sporozoite eventually may produce from 10,000 to >30,000 daughter merozoites.
   d. Both A and C
5. Which statement is true?
   a. The incubation period for S. typhi averages 1014 days but ranges from 321 days, depending on the inoculum size and the host's health and immune status
   b. The incubation period for S. typhi averages 2024 days but ranges from 721 days, depending on the inoculum size and the host's health and immune status
   c. The incubation period for S. typhi averages 21-28 days but ranges from 821 days, depending on the inoculum size and the host's health and immune status
   d. The incubation period for S. typhi averages 3014 days but ranges from 921 days, depending on the inoculum size and the host's health and immune status
6. Patient with fever, relative bradycardia, and splenomegaly with rose spots, dry cough and lymphadenopathy could be diagnosed as
   a. Chikungunya
   b. Dengue
   c. Typhoid
   d. All the above
7. Radionuclide lymphoscintigraphic imaging of the limbs reliably demonstrates widespread lymphatic abnormalities in which diseases
   a. Filariasis
   b. Plague
   c. Deep vein thrombosis
   d. Syphilis
8. Which one is correct in relation to malaria?
   a. The swollen infected liver cell eventually bursts, discharging motile merozoites into the bloodstream to produce malaria
   b. The swollen infected liver cell eventually bursts, discharging motilesporozoites into the bloodstream to produce malaria
   c. A single sporozoite eventually may produce from 10,000 to 30,000 daughtermerozoites.
   d. Both A and C

Answer

1.	b	2.	d	3.	d
4.	d	5.	a	6.	c
7.	a	8.	d		

CHAPTER 20

Pandu Roga

Introduction

One who regularly indulges in strenuous excercise, habitually take sour and salty foods, alcohol, soil and fast food, and sleep during day-hours will have one's blood vitiated by *doṣa*. This situation in turn, generate whitish yellow discolouration of the skin. The premonitory symptoms of this disease include cracking of the skin, expectoration, debility, desire for eating mud, oedema of the eye sockets (around the eyes), yellow discolouration of feces and indigestion. Varieties of *Pāṇḍu roga* includes the five types of it depending upon the causative factors viz. *vāta*, *pitta*, *kapha*, *sannipāta* (when all the three *doṣās* are simultaneously vitiated) and *Mṛtbhakṣaṇaja* intake of earth.

सर्वेषु चैतेष्विह पाण्डुभावो यतोऽधिकोऽतः खलु पाण्डुरोगः ।
(S.S.Utt. 44/4)

It is called *Pāṇḍuroga* because of predominance of paleness all over the body.

पाण्डु रोग हेतु (Pāṇḍu Roga Hetu)

क्षाराम्ललवणात्युष्णाविरुद्धासात्म्यभोजनात् ।
निष्पावमाषपिण्याकतिलतैलनिषेवणात् ॥
विदग्धेऽन्ने दिवास्वप्नाद्व्यायामान्मैथुनात्तथा ।
प्रतिकर्मर्तुवैषम्याद्वेगानां च विधारणात् ॥
कामचिन्ताभयक्रोधशोकोपहतचेतसः ।
समुदीर्णं यदा पित्तं हृदये समवस्थितम् ॥
वायुना बलिना क्षिप्तं संप्राप्य धमनीर्दश ।
प्रपन्नं केवलं देहं त्वङ्मासान्तरमाश्रितम् ॥
प्रदूष्य कफवातासृक्त्वङ्मांसानि करोति तत् ।
पाण्डुहारिद्रहरितान् वर्णान् बहुविधांस्त्वचि ॥
स पाण्डुरोग इत्युक्तः ।*(C.S.Ci. 16/7-12)*
व्यवायमम्लं लवणानि मद्यं मृदं दिवास्वप्नमतीव तीक्ष्णम् ।
निषेवमाणस्य विदूष्य रक्तं कुर्वन्ति दोषास्त्वचि पाण्डुभावम् ॥
(S.S.Utt. 44/3)

Disease *Pāṇḍu roga* manifest due to following etiological factors :

- Excess intake of alkaline, sour, saline, hot; incompatible dietetics and unwholesome food substances.
- Intake of *Niṣpāva, Māṣa, Piṇyāka,* and *Tila Taila* in excess.
- Sleeping during day time.
- Indulgence in sexual intercourse and physical exercises during indigestion.
- Erratic administration of biopurificatory therapies *(Pañcakarma).*

Faulty management of seasonal regime, suppression of natural urges particularly in persons who are afflicted with sexual desire, anxiety, fear, anger, and grief. Excess consumption of wine, eating mud, and penetrating foods.

- सन्तर्पणोत्थ विकार *(Santarpaṇottha Vikāra) C.S.Su. 23/5*
- अपतर्पणोत्थ विकार *(Apatarpaṇottha Vikāra) C.S.Su. 23/23*

पाण्डु रोग संप्राप्ति (Samprāpti of Pāṇḍu Roga)

निषेवमाणस्य विदूष्य रक्तं कुर्वन्ति दोषास्त्वचि पाण्डुभावम् ॥
(S.S.Utt. 44/3)

दोषाः पित्तप्रधानास्तु यस्य कुप्यन्ति धातुषु ।
शैथिल्यं तस्य धातूनां गौरवं चोपजायते ॥
ततो वर्णबलस्नेहा ये चान्येऽप्योजसो गुणाः।
व्रजन्ति क्षयमत्यर्थं दोषदूष्यप्रदूषणात् ॥
सोऽल्परक्तोऽल्पमेदस्को निःसारः शिथिलेन्द्रियः ।
वैवर्ण्यं भजते, तस्य हेतुं शृणु सलक्षणम् ॥
(C.S.Ci. 16/4-6)

समुदीर्णं यदा पित्तं हृदये समवस्थितम् ॥
वायुना बलिना क्षिप्तं संप्राप्य धमनीर्दश ।
प्रपन्नं केवलं देहं त्वङ्मासान्तरमाश्रितम् ॥
प्रदूष्य कफवातासृक्त्वङ्मांसानि करोति तत् ।
पाण्डुहारिद्रहरितान् वर्णान् बहुविधांस्त्वचि ॥
स पाण्डुरोग इत्युक्तः । *C.S.Ci. 16/9-12*

Due to consumption of etiological factors *Doṣa* aggravates especially *Pitta doṣa* and afflicts *dhātus* mainly *Raktadhātu* as a result it brings laxity and heaviness in the *Dhātus*. Heaviness of *Dhātus* manifest due to the disturbance of normal functions of *Dhātus*. Patients of *Pāṇḍu roga* loses their vitality, complexion, strength, unctuousness and other properties of *Ojas* due to morbidity of *Doṣa* and *Dūṣya*. That's why patients of *Pāṇḍu Roga* suffers from deficiency of *Rakta, Meda, Niḥsāra* (loss of essence) associated with improper functioning of sense organs and discolouration.

Aggravated *Pitta* expelled from *Hṛdaya Via* ten blood vessels, by powerful *Vāta* circulates all over the body. It reaches the space between *Tvak* and *Māmsa* and brings abnormality in *Kapha, Vāta, Rakta, Tvak* and *Māmsa* leading to abnormal complexion like pale yellow, deep yellow and greenish colouration.

Aggravated *Pitta* is responsible for the less production of *Poṣaka* (nutrient portion) from the *Rasadhātu* as a result depletion of *Rakta* takes place.

Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Pittādi doṣās*

↓

Diminution of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Aggravated *Pitta* expelled from *Hṛdaya Via* ten blood vessels, by powerful *Vāta* circulates all over the body (afflicts *dhātus* mainly *Raktadhātu*) and it is also responsible for the less production of *Poṣaka* (nutrient portion) from the *Rasadhātu* as a result depletion of *Rakta* takes place

↓

It reaches the space between *Tvak* and *Māmsa*

↓

Brings laxity and heaviness in the *Kapha, Vāta, Rakta, Tvak* and *Māmsa*

↓

leading to abnormal complexion like pale yellow, deep yellow and greenish colouration.

↓

deficiency of *Rakta, Meda, Niḥsāra* (loss of essence) associated with improper functioning of sense organs and discolouration.

↓

Pāṇḍu roga

(Patients of *Pāṇḍu roga* loses their vitality, complexion, strength, unctuousness and other properties of *Ojas* due to morbidity of *Doṣa* and *Dūṣya*)

पाण्डु रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Pāṇḍu Roga)

Doṣa	: *Pitta Pradhāna Tridoṣaja* *Pitta → Sādhaka, Rañjaka & Bhrājaka* *Kapha → Avalambaka, Kledaka* *Vāta → Vyānavāta*
Dūṣya	: *Dhātu → Rasa, Rakta, Māmsa, Meda;* later all the *Dhātus (Nihsāra)* *Upadhātu → Sarvaupadhātu* *Śārīrika Mala → Mūtra, Puriṣa* *Dhātu Mala → Kapha, Pitta*
Agni	: *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi	: *Mandāgni*
Āma	: *Āgnijanya*
Srotas	: *Rasavaha, Raktavaha*
Srotoduṣṭi	: *Saṅga, Vimārgagamana*
Udbhavasthāna	: *Āmāśayottha*
Sancārasthāna	: *Tvak & Māmsa*
Vyaktasthāna	: *Tvak*
Svabhāva	: *Cirakāri*
Prabhāva	: *Mentioned under prognosis*
Roga bheda	: 05 types *(Caraka)* 04 types *(Suśruta)*

पूर्वरूप (Pūrvarupa)

त्वक्स्फोटनं ष्ठीवनगात्रसादौ मृद्भक्षणं प्रेक्षणकूटशोथः ।
विण्मूत्रपीतत्वमथाविपाको भविष्यतस्तस्य पुरःसराणि ॥
(S.S.Utt. 44/5)

तस्य लिङ्गं भविज्यतः।
हृदयस्पन्दनं रौक्ष्यं स्वेदाभावः श्रमस्तथा॥

(*C.S.Ci. 16/12*)

Premonitory signs and symptoms of *Pāṇḍu roga* are as follows :- Cracking of the skin, splitting of sputum, general body malaise, liking for mud intake, swelling over eye socket, yellowish discolouration of faecal matter and urine; indigestion, palpitation, roughness, absence of perspiration and exhaustion.

सामान्य लक्षण (Sāmānya Lakṣaṇa)

संभूतेऽस्मिन् भवेत् सर्वः कर्णक्ष्वेडी हतानलः।
दुर्बलः सदनोऽन्नद्विट् श्रमभ्रमनिपीडितः॥
गात्रशूलज्वरश्वासगौरवारुचिमान्नरः।
मृदितैरिव गात्रैश्च पीडितोन्मथितैरिव॥
शूनाक्षिकूटो हरितः शीर्णलोमा हतप्रभ;।
कोपनः शिशिरद्वेषी निद्रालुः ष्ठीवनोऽल्पवाक्॥
पिण्डकोद्वेष्टकट्यूरुपादरुक्सदनानि च।
भवन्त्यारोहणायासैर्विशेषश्चास्य वक्ष्यते॥

(*C.S.Ci. 16/13-16*)

General signs and symptoms of *Pāṇḍuroga* are as follows:-

- Tinnitus, loss of functions of *Agni,* debility, general malaise, aversion towards food, fatigue, giddiness, pain all over the body, fever, breathlessness, heaviness and anorexia.
- Patient feels as if his limbs are being kneaded, pressed or churned.
- Swelling over orbit; complexion become greenish; hairfall; loss of lustre; irritability; dislikes cold things; sleepy always; spitting; speaks little; cramps in the calf muscles; patients suffers from pain in waist, thighs and legs; while climbing stairs patient feels exhausted.

पाण्डु रोग भेद (Pāṇḍu Roga Bheda)

Pāṇḍu roga is classified into 5 types in *Caraka Saṁhita* and 4 types in *Suśruta Saṁhita* are as follows:-

पाण्डुरोगाः स्मष्टताः पञ्च वातपित्तकफैस्त्रयः।
चतुर्थः सन्निपातेन पञ्चमो भक्षणान्मष्टदः॥

(*C.S.Ci. 16/3*)

पाण्ड्वामयोऽष्टार्द्धविधः प्रदिष्टः पृथक् समस्तैर्युगपच्च दोषैः।

(*C.S.Utt. 44/4*)

- *Vātaja Pāṇḍu Roga*
- *Pittaja Pāṇḍu Roga*
- *Kaphaja Pāṇḍu Roga*
- *Sannipātaja Paṇḍu Roga*
- *Mṛttikā Bhakṣaṇa Janya Pāṇḍu Roga*

(Others feel that *Mṛttikā bhakṣaṇa Janya Pāṇḍu roga* was included under *Sannipātaja Pāṇḍu roga* in *Suśruta Samhita*)

वातज पाण्डु रोग हेतु, संप्राप्ति एवं लक्षण (*Vātaja Pāṇḍu Roga Hetu, Samprāpti Evam Lakṣaṇa*)

आहारैरुपचारैश्च वातलैः कुपितोऽनिलः।
जनयेत्कृष्णपाण्डुत्वं तथा रूक्षारुणाङ्गताम्॥
अङ्गमर्द रुजं तोदं कम्पं पार्श्वशिरोरुजम्।
वर्चःशोषास्यवैरस्यशोफानाहबलक्षयान्॥

(*C.S.Ci. 16/17-18*)

कृष्णोक्षणं कृष्णसिराऽवनद्धं तद्वर्णविण्मूत्रनखाननञ्च।
वातेन पाण्डुं मनुजं व्यवस्येद् युक्तं तथाऽन्यैस्तदुपद्रवैश्च॥

(*S.S.Utt. 44/7*)

Hetu evam Samprāpti : Due to consumption of *Vāta* excacerbating dietetics and activities leads to aggravation of *Vāta* and manifest *Vātaja Pāṇḍuroga.*

Lakṣaṇa : Blackish discolouration, Pallor, roughness, reddishness of the body, general malaise, discomfort, pricking pain, tremors, pain in sides and head, dryness of stool, distaste in oral cavity, oedema, constipation and loss of strength. Blackish discolouration of eyes, blackish network of blood vessels all over the body, blackish discolouration of stool, urine, nails and oral cavity and other symptoms of *Vātaja* aggravation.

पित्तज पाण्डु रोग हेतु, संप्राप्ति एवं लक्षण (*Pittaja Pāṇḍu Roga Hetu, Samprāpti Evam Lakṣaṇa*)

पित्तलस्याचितं पित्तं यथोक्तैःस्वैः प्रकोपणैः।
दूषयित्वा तु रक्तादीन् पाण्डुरोगाय कल्पते॥
स पीतो हरिताभो वा ज्वरदाहसमन्वितः।
छर्दिमूर्च्छापिपासार्तः पीतमूत्रशकृन्नरः॥
स्वेदनः शीतकामश्च न चान्नमभिनन्दति।
कटुकास्यो न चास्योष्णमुपशेतेऽम्लमेव च॥
उद्गारोऽम्लो विदाहश्च विदग्धेऽन्नेऽस्य जायते।

दौर्गन्ध्यं भिन्नवर्चस्त्वं दौर्बल्यं तम एव च ॥

(C.S.Ci. 16/19-22)

पीतेक्षणं पीतसिराऽवनद्धं तद्वर्णविण्मूत्रनखाननञ्च ।
पित्तेन पाण्डुं मनुजं व्यवस्येद् युक्तं तथाऽन्यैस्तदुपद्रवैश्च ॥

(S.S.Utt. 44/8)

Pitta is already disturbed in *Pāṇḍu Roga*, in such circumstances if patient again consumes *Pitta* aggravating dietetics and behaviour. If *Pitta prakṛti* person indulges dietetics and activities which excacerbates *Pitta*.

Samprāpti : Due to intake of *Nidāna, Pitta* aggravates in excess and brings abnormality in *Rakta* and manifest *Pittaja Pāṇḍuroga*.

Lakṣaṇa : Complexion becomes either yellow or green, fever, burning sensation, faints due to excessive thirst, morbid thirst, yellowish discolouration of stool and urine, profuse perspiration, desires to take cold substances, aversion towards food, mouth taste becomes pungent, uncomfortable to hot & sour things, sour eructations, patient feels burning sensation during digestion of food, foul smell from oral cavity. *Diarrhoea*, debility and feeling of darkness infront of the eye. Yellowish discolouration in eyes, yellowish network of veins appear all over the body. Yellowish discolouration of skin, faeces, urine, nails & oral cavity.

कफज पाण्डु हेतु, संप्राप्ति एवं लक्षण (Kaphaja Pāṇḍu Hetu, Samprāpti Evam Lakṣaṇa)

विवृद्धः श्लेष्मलैः श्लेष्मा पाण्डुरोगं स पूर्ववत् ।
करोति गौरवं तन्द्रां छर्दिं श्वेतावभासताम् ॥
प्रसेकं लोमहर्षं च सादं मूर्च्छां भ्रमं क्लमम् ।
श्वासं कासं तथाऽऽलस्यमरुचिं वाक्स्वरग्रहम् ॥
शुक्लमूत्राक्षिवर्चस्त्वं कटुरूक्षोष्णकामताम् ।
श्वयथुं मधुरास्यत्वमिति पाण्ड्वामयः कफात् ॥

(C.S.Ci. 16/23-25)

शुक्लेक्षणं शुक्लसिराऽवनद्धं तद्वर्णविण्मूत्रनखाननञ्च ।
कफेन पाण्डुं मनुजं व्यवस्येद् युक्तं तथाऽन्यैस्तदुपद्रवैश्च ॥

(S.S.Utt. 44/9)

Etiological factors, pathogenesis and symptomatology of *Kaphaja Pāṇḍu Roga* are as follows:-

***Hetu & Samprāpti* :** Excessive consumption of *Kapha* provocative dietetics and behaviour leads to development of *Kaphaja Pāṇḍu Roga*.

Lakṣaṇa : Heaviness, drowsiness, vomiting, complexion becomes white, salivation in excess, horripilation, general malaise, fainting, giddiness, mental fatigue, breathlessness, cough, laziness, anorexia, loss of speech and voice, whitish discolouration observed in eyes, nail, urine, and stool; likes to take pungent substances, rough and hot things; oedema sweetishness in oral cavity, whitish network of blood vessels seen all over the body and other *Kapha* aggravating symptoms.

सन्निपातज पाण्डुरोग हेतु, संप्राप्ति एवं लक्षण (Sannipātaja Pāṇḍuroga hetu, Samprāpti Evam Lakṣaṇa)

सर्वात्मके सर्वमिदं व्यवस्येद ।
वक्ष्यामि लिङ्गान्यथ कामलायाः ॥

(S.S.Utt. 44/10)

सर्वान्नसेविनः सर्वे दुष्टा दोषास्त्रिदोषजम् ।
त्रिदोषलिङ्गं कुर्वन्ति पाण्डुरोगं सुदुःसहम् ॥

(C.S.Ci. 16/26)

***Hetu evam Samprāpti* :** If person consumes all kinds of food substances without following rules and regulations of dietetics leading to excacerbation of all the three *Doṣās* and manifest *Sannipātaja Pāṇḍu roga*.

***Lakṣaṇa* :** All the symptomatology of *Tridoṣa* appears in this disease and it is extremely intolerable because it develops complications.

मृत्तिका भक्षणजन्य पाण्डुरोग हेतु, संप्राप्ति एवं लक्षण (Mṛttikā Bhakṣaṇajanya Pāṇḍu roga Hetu, Samprāpti Evam Lakṣaṇa)

मृत्तिकादनशीलस्य कुप्यत्यन्यतमो मलः ।
कषाया मारुतं, पित्तमूषरा, मधुरा कफम् ॥
कोपयेन्मृद्रसादींश्च रौक्ष्याद्भुक्तं विरूक्षयेत् ।
पूरयत्यविपक्वैव स्रोतांसि निरुणद्धि च ॥
इन्द्रियाणां बलं हत्वा तेजो वीर्यौजसी तथा ।
पाण्डुरोगं करोत्याशु बलवर्णाग्निनाशनम् ॥
शूनगण्डाक्षिकूटभ्रूः शूनपान्नाभिमेहनः ।
क्रिमिकोष्ठोऽतिसार्येत मलं सासृक् कफान्वितम् ॥

(C.S.Ci. 16/27-30)

One who consumes mud (clay) habitually causes aggravation of either *Vāta* or *Pitta* or *Kapha*.

Astringent taste of mud aggravates *Vāta*, alkaline taste of mud aggravates *Pitta* and sweet taste of mud aggravates *Kapha*. Due to its ununctuousness it brings dryness in the *Rasādhi dhātus*. Undigested mud fills up channels and brings obstruction in them as a result loss of functions of senses, strength, lustre, energy and *Ojas*. This type of *Pāṇḍu Roga* further deteriorates strength, complexion and power of *Agni*.

Lakṣaṇa :

Clinical features of *Mṛttikā Bhakṣaṇajanya Pāṇḍu roga* are as follows:- Oedema around cheek, eye sockets and eyebrows; oedema in the feet, umbilical region and genital parts; development of intestinal worms; diarrhoea associated with blood and mucus.

उपद्रव (Upadrava)

उपद्रवास्तेष्वरुचिः पिपासा छर्दिर्ज्वरो मूर्धरुजाऽग्निसादः।
शोफस्तथा कण्ठगतोऽबलत्वं मूर्च्छा क्लमो हृद्यवपीडनं च॥
(S.S.Utt. 44/13)

The following complications arise in *Pāṇḍuroga*. Loss of taste, thirst, vomiting, fever, headache, sluggishness in the functions of agni, swelling in the throat, debility, fainting, exhaustion and discomfort in the region of heart.

साध्यासाध्यता (Sādhyāsādhyatā)

अन्तेषु शूनं परिहीनमध्यं म्लानं तथाऽन्तेषु च मध्यशूनम्।
गुदे च शेफस्यथ मुष्कशूनं प्रताम्यमानं च विसंज्ञकल्पम्॥
विवर्जयेत् पाण्डुकिनं यशोर्थी तथाऽतिसारज्वरपीडितञ्च॥
(S.S.Utt. 44/43-44)

Physician should reject the patient of *Pāṇḍuroga* for management if patient is suffering from swelling in the extremities and emaciation in the trunk; or swelling observed in both extremities, trunk. anus, penis, and scroatum; one who faints often; loses consciousness and suffering from diarrhoea and fever.

पाण्डुरोगश्चिरोत्पन्नः खरीभूतो न सिध्यति।
कालप्रकर्षाच्छूनो ना यश्च पीतानि पश्यति॥
बद्धाल्पविट्कं सकफं हरितं योऽतिसार्यते।
दीनः श्वेतातिदिग्धाङ्गश्छर्दिमूर्च्छातृषार्दितः॥
स नास्त्यसृक्क्षयाद्यश्च पाण्डुः श्वेतत्वमाप्नुयात्।
इति पञ्चविधस्योक्तं पाण्डुरोगस्य लक्षणम्॥
(C.S.Ci. 16/31-33)

The following points indicate incurability of *Pāṇḍu Roga*:-

- Persistence of disease for longer duration associated with excessive roughness.
- Due to chronicity patient develops oedema. Patients visualises the objects as yellow.
- Patients will be suffering from either constipation or scanty stool. Patient passes loose stools associated with mucus having greenish discolouration. Having dull look; excessive paleness as if he is smeared; patients suffers from severe trouble due to excessive vomiting, fainting and morbid thirst. If patient develops severe paleness due to deficiency of blood.

उपशय/पथ्य (Upaśaya/Pathya)

C.S.Ci. 16th Chapter

S.S.Utt. 44th Chapter

शालीन् सयवगोधूमान् पुराणान् यूषसंहितान्॥
मुद्गाढकीमसूरैश्च जाङ्गलैश्च रसैर्हितैः।
यथादोषं विशिष्टं च तयोर्भैषज्यमाचरेत्॥
पञ्चगव्यं महातिक्तं कल्याणकमथापि वा।
स्नेहनार्थं घृतं दद्यात् कामलापाण्डुरोगिणे॥
(C.S.Ci. 16/41-43)

Samśodhana therapy *(Vamana, Virecana)*, *Śālianna*, barley, wheat mixed with *Yūṣa* prepared from *Mudga*, *Āḍhaki* and *Masūra;* and meat soup of wild animals and birds. Specific medicines are to be administered depending on the predominance of *Doṣās*. *Pañcagavya ghṛta*, *Mahātikta ghṛta* and *Kalyāṇaka ghṛta* are useful for the purpose of oleation therapy.

One who is suffering from *Pāṇḍuroga* should use *Ariṣṭa* prepared from *Guḍa*, honey and *Sugar; Āsava* prepared from urines and *Kṣārās;* meat soup added with fats and *Āmalaka* juice *(S.S.Utt. 44/36-37)*

Dāḍimādya ghṛta
Kaṭukādya ghṛta
Drākṣā ghṛta
Navāyasa Cūrna
Yogarāja
Śilājatu Vaṭaka
Dāryādi Leha
Gauḍāriṣṭa
Dhātryariṣṭa
Pathya ghṛta
Danti ghṛta
Haridrādi ghṛta
Maṇḍura vaṭaka
Tāpyādi yoga
Punarnavā Maṇḍūra
Dhātravaleha
Bijakāriṣṭa
Vyoṣādi ghṛta

The following measures are helpful for the patient of *Pāṇḍuroga* : *Vamana* (vomiting), *Virecana* (purgation) and processing the burning therapy through below the naval, in the forehead, in the bottom of the hands, in the joints of the legs, in the armpits and around the middle of the breast. Similarly, the patient may take the following edibles: old barley, wheat, *Śāli* variety of rice, *Mudga*, pigeon peas, lentils, flash and meat juice of wild animals and birds, *Paṭola*, ripe ash gourd, unripe bananas, *Jīvantī* leaves, *Yavakṣāra*, *Guḍūcī*, *Taṇḍulīkaya* leaves, *Punarnavā*, *Droṇapuṣpī*, aubergine, garlic, onion, ripe mango, *Haraḍa*, *Bimbi*, *Śṛīngī* fish, cow-urine, *Āmalakī* fruits, butter milk, *Ghṛta*, oil, *Sauvīra*, *Tuṣodaka* (rice-washing), butter, sandalwoods, *Haridrā*, and *Keśara*. (B.R. 12/147-152)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Avoid etiological factors.

Blood-letting, smoking, sweating and intercourse are to be avoided by the patient of *Pāṇḍu Roga*:- Similarly he should avoid consumption of peas, cooked leaves, asafoetida, *Urada* peas, sesame-cakes, betel leaves, liquor, red chilies, all types of salt, sour edibles, incompatible foods, food items that are heavy to digest and cause burning sensation, etc. Intake of water of rivers like those originating from *Vindhyā* and *Sahyādri* mountains, staying around fire, around sun, hard works and exercise, anger, suppression of natural urges and sleeping during day time is prohibited in *Pāṇḍuroga*. (B.R. 12/153-155)

Differentiate doṣaja Pāṇḍu

S.No.	***Vātaja Pāṇḍu Roga***	***Pittaja Pāṇḍu Roga***	***Kaphaja Pāṇḍu Roga***	***Sannipātaja Pāṇḍu Roga***	***Mṛttikā Bhakṣaṇa Janya Pāṇḍu Roga***
Etiology	Due to consumption of *Vāta* exacerbating dietetics and activities	*Pitta* is already disturbed in *Pāṇḍu Roga*, in such circumstances if patient again consumes *Pitta* aggravating dietetics and behavior. If *Pitta prakṛti* person indulges dietetics and activities which exacerbates *Pitta*	Excessive consumption of *Kapha* provocative dietetics and behaviour leads to development of *Kaphaja Pāṇḍu Roga*	If person consumes all kinds of food substances without following rules and regulations of dietetics	One who consumes mud (clay) habitually causes aggravation of either *Vāta* or *Pitta* or *Kapha*. Astringent taste of mud aggravates *Vāta*, alkaline taste of mud aggravates *Pitta* and sweet taste of mud aggravates *Kapha*.
Clinical presentations	Blackish discoloration of eyes, blackish network of blood vessels all over the body, blackish discoloration of stool, urine, nails and oral cavity	Yellowish discolouration in eyes, yellowish network of veins appear all over the body. Yellowish discolouration of skin, faeces, urine, nails & oral cavity.	Whitish discoloration observed in eyes, nail, urine, and stool & whitish network of blood vessels seen all over the body	All the symptomatology of *Tridoṣa* appears in this disease and it is extremely intolerable because it develops complications	Oedema around cheek, eye sockets and eyebrows; oedema in the feet, umbilical region and genital parts; development of intestinal worms; diarrhoea associated with blood and mucus.
Modern correlation	Nutritional deficiency anaemia	Haemolyitc anaemia & megaloblastic anaemia	Megaloblastic anaemia	Anaemia of chronic diseases llike malaria, typhoid, rheumatoid arthritis	Anaemia due to worm infestations folate & vitamin B12 deficiency

MCQs

1. *Shoonanabhipadaasyamehana* is seen in which disease
   a. *Svayathu* b. *Mritbhakshanajanyapandu*
   c. *Udara* d. *Krimi*
2. Types of *Pandu* as per *Sushruta*
   a. 5 b. 4 c. 3 d. 6
3. If abnormal *Kapha* enters *Raktadhatu* manifest which diseases?
   a. *Vishpota* b. *Masoorika*
   c. *Visarpa* d. *Panduroga*
4. *Dushya* in *Panduroga* as per *Sushruta*
   a. *Twak Rakta* b. *Rakta*
   c. *Asruk Mamsa* d. *Twak Rakta Mamsa*
5. *Hrdayaspandana* seen in
   a. *Pandupurvarupa*
   b. *Vatajapandu*
   c. *Kaphajapandu*
   d. *Mrtbhakshanajanyapandu*
6. *Sheernaloma hataprabha* is the *lakshana* of
   a. *Haleemaka* b. *Kamala*
   c. *Pandu* d. *Visarpa*
7. *Lavanavaktratwam is the feature of*
   a. *Pittajapandu* b. *Kaphajapandu*
   c. *Kaphajasopha* d. *Kaphajavisarpa*
8. *Hatendriya is the feature of*
   a. *Vatajapandu*
   b. *Kaphajapandu*
   c. *Mritbhakshanajanyapandu*
   d. *Tridoshaja Pandu*
9. *Antheshushoonampariheenamadyam* is the *Asadya lakshana of*
   a. *Gulma* b. *Pandu* c. *Udara* d. *Arsas*
10. *What is the prognosis of Pandu with Atisara and Jwara*
    a. *Krichrasadya* b. *Yapya*
    c. *Asadya* d. *Krichrasadya tama*
11. *Indriyanam balam hatwa tejo viryoujasi tatha*
    a. *Vatajapandu* b. *Kaphajapandu*
    c. *Mritbhakshanajanya pandu*
    d. *Tridoshaja Pandu*

Answer

1.	b	2.	b	3.	d
4.	c	5.	a	6.	c
7.	b	8.	d	9.	b
10.	c	11.	c		

CHAPTER

21

Rakta-Pitta

Introduction

Bleeding disorders. It is an indisposition in which bleeding occurs from the upper channels like nose, mouth etc. or from lower channels like anus, urinary passage, female genital organ etc. Bleeding from the upper channels like nose, mouth etc. or from lower channels like anus, urinary passage, female genital organ, etc. or petechial haemorrhage, aesthania, cough, dyspnoea, fever, loss of appetite, indigestion, vomiting, thirst, burning sensation, nervousness, frequent motions, pallor and perspiration are the main signs and symptoms of *Raktapitta*. Overexposure to the sun, excessive physical exercise, grief, travelling long distances on foot and sexual intercourse, as also excessive use of sharp, hot, alkaline, saltish, acidic and pungent substances all these factors vitiate the *pitta*. The vitiated *pitta* in turn provoke *rakta* and causes it to flow out of the body through upper and lower orifices.

The upper orfices in the context include the nostrils, eyes, ears and mouth while the urethra, vagina, rectum are the lower passages. In the disease *raktapitta* the blood comes out from these passages and if the ailment has attained severe conditions, blood might come out of the hair follicles of the skin also. By excessive exposure to sun, excessive indulgence in exercise, excessive grief, excessive walk, excessive indulgence in sex and excessive intake of sharp, hot, alkaline, saline, sour and pungent food, *pitta* gets vitiated (*vidagdha*), and by its own attributes vitiates blood. As a result of this, bleeding takes place through upward tracts, viz. nose, eyes, ears and mouth or downward tracts, viz. urethra, vagina and anus or through both tracts including hair-roots. Prostration, desire for cold Haemoptysis., smoky feeling in throat, vomiting and breath having smell of iron, these are the premonitory signs and symptoms of *rakta pitta*.

In *ślaiṣmika* type of *rakta pitta* the blood becomes thick (more density) pale yellow, unctuous and slimy. The blood in *vātika* type of *rakta pitta* is grayish black or reddish in colour, foamy thin and unctuous. In *paittika* type of *rakta pitta*, the blood looks like the flower of *Pāṭala* or black or like the urine of the cow on greasy-black or house shut or collyrium. In *sannipātika* type of *rakta pitta*, signs of all the three types, viz. *vātika*, *paittika* and *ślaiṣmika* appears in the blood. *Rakta pitta* having upward direction gets mixed up with *kapha*, the one having downward direction gets mixed up with *vāyu* and the variety of *rakta pitta* moving in both the directions i. e. upwards and downwards gets mixed up with both *kapha* and *vāyu*.

The *rakta pitta* of upward direction is curable, of downward direction is palliable and of both the upward and downward directions is incurable. By the habitual intake of unwholesome food, the polluted *rasa* vitiates *pitta* and moves either upwards from the stomach or downwards from the colon. When *rasa* in both the stomach and colon is vitiated, bleeding takes place from both the directions. Weakness, dyspnoea, cough, fever, vomiting, feeling of intoxication, pallor, burning sensation, fainting, serious type of burning sensation after food, loss of patience, pain like that of heart disease, morbid thirst, diarrhoea, headache, spitting of purified material, disliking for food and indigestion-these are the complications of *rakta pitta*. If the blood is like the water in which meat is washed, if it looks as if boiled, if it appears like mud, water, fat, pus, lymph, pieces of liver or fruit of *jambū*, if it is black or blue in colour if it smells extremely foul like a dead body and if it looks like a rainbow, then the patient of *rakta pitta* is incurable. If to a patient of *rakta pitta*, the sky appears red like blood, if he frequently vomits blood, if his eyes become blood-red and if his eructaions are mixed with blood then such a patient undoubtedly succumbs to death. If there is a great excacerbation of *doṣās* and the patient is not suffering from loss of strength, muscles & digestive fire, then fasting therapy may be advised.

Ban. *raktpitā*, Guj. *raktpitā*, Hin. *raktpitā*, Kan. *rakthāriyavudū*, Mal. *rkth& pitām*, Mar. *raktpitā*, Ori. *raktpitā*, Tam. *pulngglilrthth akāśīvū*.

संसर्गाल्लोहितप्रदूषणाल्लोहितगन्धवर्णानुविधानाच्च पित्तं लोहित- पित्तमित्याचक्षत ॥ *C.S.Ni. 2/5*

संयोगाद्दूषणात्तत्तु सामान्याद्गन्धवर्णयोः ।
रक्तस्य पित्तमाख्यातं रक्तपित्तं मनीषिभिः ॥
C.S.Ci. 4/9

Pitta is called *Raktapitta* because of its nature to amalgamate with *rakta* and later it acquires the smell and colour.

रक्तपित्त हेतु (Rakta-Pitta Hetu)

यदा जन्तुर्यवकोद्दालककोरदूषप्रायाण्यन्नानि भुङ्क्ते, भृशोष्णतीक्ष्णमपि चान्यदन्नजातं निष्पावमाषकुलत्थसूपक्षारोपसंहितं, दधिदधिमण्डोदश्वित्कट्वराम्लकाञ्जिकोपसेकं वा, वाराहमाहिषाविकमात्स्यगव्यपिशितं, पिण्याकपिण्डालुशुष्कशाकोपहितं, मूलकसर्षपलशुनकरञ्जशिग्रुमधुशिग्रु (खडयूष); भूस्तृणसुमुखसुरसकुठेरकगण्डीरकालमालकपर्णासक्षवकफणिज्झकोपदंश, सुरासौवीरतुषोदकमैरेयमेदकमधूलकशुक्तकुवलबदराम्लप्रायानुपानं वा, पिष्टान्नोतरभूयिष्ठम्; उष्णाभितप्तो वाऽतिमात्रमतिवेलं वाऽऽमं पयः पिबति, पयसा समश्नाति रोहिणीकं, काणकपोतं वा सर्षपतैलक्षारसिद्धं, कुलत्थपिण्याकजाम्ववलकुचपक्वै : शौक्तिकैर्वा सह क्षीरं पिबत्युष्णाभितप्तः । *C.S.Ni. 2/4*

Causative factors for the manifestation of *Rakta-Pitta* are described below : Excess consumption of *Yavaka, Uddālaka, Koradūṣa,* intake of hot and irritating foods added with *Niṣpāva, Māṣa, Kulattha, Kṣāra,* or with curd, curd water, butter milk, sour butter milk or sour gruel. Excess intake of meat of Pig, Buffalow, Sheep, Fish and Cow, Oil cake, *Piṇḍālu* and dried fat herbs or dry vegetables. Salad or pickles comprising of Radish, Mustard, Garlic, *Karañja, Śigru, Madhu śigru, Khaḍayuṣa, Bhūstṛṇa, Sumukha, Surasa, Kuṭheraka, Gaṇḍīra, Kāla mālaka, Parṇāsa, Kṣavaka, Phaṇijjhaka* or followed by *Surā, Sauvīra, Tuṣodaka, Mairey medaka, Madhūlaka, Sukta, Sour* preparations of *Kuvala* and *Badara;* intake of excess pastries after food; frequent or excess intake of milk after exposure to heat; intake of milk with *Rohiṇika* vegetable; *Kaṇakapota* (a type of pigeon) boiled with mustard oil or alkalies; consumption of milk with *Kulattha,* Oil cake, fruits of *Jambu* and *Lakuca*.

क्रोधशोकभयायासविरुद्धान्नातपान्नन् । कट्वम्ललवणक्षारतीक्ष्णोष्णातिविदाहिनः ॥ नित्यमभ्यसते॥

S.S.Utt. 45/3-4

Excessive indulgence in anger, sorrow, fear, exertion, incompatible foods, excessive exposure to sunlight and fire; excess intake of pungent, sour, salt, alkaline, penetrating, hot, and food substances which induce burning sensation during digestion.

रक्तपित्तप्रकोपस्तु खलु पुरा दक्षयज्ञोद्ध्वंसे रुद्रकोपामर्षाग्नि प्राणिनां परिगतशीररप्राणानामभवज्ज्वरमनु॥*C.S.Ni. 2/10*

During early times Lord *Rudra* was born to destruct *Dakṣās* sacrifice by the anger of *Lord Śiva.* After destructing sacrifice, *Rudra* came to earth and suffering the living beings in the form of *Jvara* and *Rakta-Pitta.*

रक्तपित्त संप्राप्ति (Samprāpti of Rakta-Pitta)

तस्यैवमाचरतःपित्तंप्रकोपमापद्यते, लोहितं च स्वप्रमाणमतिवर्तते। तस्मिन् प्रमाणातिवृत्ते पित्तं प्रकुपितं शरीरमनुसर्पद्यदेवयकृत्प्लीहप्रभावानांलोहितवहानां च स्त्रोतसां लोहिताभिष्यन्दगुरूणि मुखान्यासाद्य प्रतिरुन्धयात् तदेव लोहितं दूषयति । *C.S.Ni. 2/4*

दुष्टो रसः पित्तं प्रकोपयेत् । विदग्धं स्वगुणैः पित्तं विदहत्याशु शोणितम् ॥ ततः प्रवर्त्तते रक्तमूर्ध्वं चाधो द्विधाऽपि वा ॥

S.S.Utt. 45/4-5

तैर्हेतुभिः समुत्क्लिष्टं पित्तं रक्तं प्रपद्यते ।
तद्योनित्वात् प्रपन्नं च वर्धते तत् प्रदूषयत् ॥
तस्योष्मणा द्रवो धातुर्धातोर्धातोः प्रसिच्यते ।
स्विद्यतस्तेन संवृद्धिं भूयस्तदधिगच्छति ॥
C.S.Ci. 4/7-8

Due to consumption of etiological factors mentioned above *Pitta* gets aggravated and *Rakta* exceeds its normal quantity. Due to excess increase in the quantity of *Rakta* leads to opening of *Lohitavaha Srotas* which originate from spleen and liver. *Pitta* takes origin from *Rakta.* Excacerbated *Pitta* enters *Śoṇitavaha Srotas* and obstruct them resulting in vitiation of *Rakta. Pitta* after amalgamating with *Rakta* gets further aggravated. Liquid fraction of *Pitta* pervades one *Dhātu* after other. Due to the effect of heat of *Pitta* excessive liquid exudate from all the *Dhātus*. These liquids after mixing with *Pitta* leads to excessive aggravation or increase in quantity of *Pitta* as a result of which *Rakta (pitta)* comes out of the body through either upper or lower orifices or both together. It is caused by the vitiation of *Rakta* by *Pitta*.

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Pittādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Aggravation of *Pitta* and *Rakta* exceeds its normal quantity
↓
Opening of *Lohitavaha Srotas* due to excessive *Rakta*
↓
Excacerbated *Pitta* enters *Śoṇitavaha Srotas and* amalgamate with *Rakta* gets further aggravated
↓
Liquid fraction of *Pitta* pervades one *Dhātu* after other
↓
↓ Due to the effect of heat of *Pitta* excessive liquid exudate from all the *Dhātus*
↓
↓These liquids after mixing with *Pitta* leads to excessive aggravation or increase in quantity of *Pitta*
↓
As a result of which *Rakta (pitta)* comes out of the body through either upper or lower orifices or both together.
↓
Rakta-Pitta.
(It is caused by the vitiation of *Rakta* by *Pitta*)

रक्तपित्त संप्राप्ति घटक (Samprāpti Ghaṭaka of Rakta-Pitta)

Doṣa : *Pitta Pradhāna Tridoṣaja*
Vāta → Samāna, Apāna, Vyāna
Pitta → Pācaka
Kapha → Kledaka
Dūṣya *Dhātu → Rakta, Rasa, Māmsa*
Updhātu → Sirā
Śārīrika Mala → Mūtra, Puriṣa
Dhātu Mala → Pitta, Kapha
Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Agnijanya*
Srotas : *Raktavaha, Rasavaha, Annavaha*
Srotoduṣṭi : *Saṅga, Vimārgagamana*
Adhiṣṭhāna : *Yakṛt & Plīhā*
Udbhavasthāna : *Āmāśaya, Raktavaha Srotomūla*
Roga Mārga : *Bāhya & Abhyantara*
Vyaktasthāna : *Ūrdhva & Adhogata bāhya srotas, (Nāsa, Karṇa, Meḍhra, Yoni, guda, Romakupa)*
Rogabheda : 3 types *Ūrdhvaga, Adhoga, Ubhaya*
Sancārasthāna : *Rasavāhinyaḥ, Raktavāhinyaḥ*
Svabhāva : *Aśukāri, Chirakāri*
Prabhāva : General Prognosis rule

Location of *Raktapitta*

प्लीहानं च यकृच्चैव तदधिष्ठाय वर्तते ।
स्रोतांसि रक्तवाहिनि तन्मूलानिहि देहिनाम ॥
C.S.Ci. 4/10

Disease *Raktapitta* located in spleen and liver, and from there it goes upwards and downwards. Liver and spleen are the root for the *Raktavaha Srotas*.

पूर्वरूप (Pūrvarūpa)

तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा-अनन्नाभिलाषः, भुक्तस्य विदाहः, शुक्ताम्लगन्धरस उद्गारः, छर्देरभीक्ष्णमागमनं, छर्दितस्य बीभत्सता, स्वरभेदो, गात्राणां सदनं, परिदाहः, मुखाद्धूमागम इवं लोहलोहितमत्स्यामगन्धित्वमिव चास्यस्य, रक्तहरितहारिद्रत्वमङ्गावयशकृन्मूत्रस्वेदलालासिङ्घाणकास्यकर्णमलपिडकोलिकापिडकानाम्, अङ्गवेदना, लोहितनीलपीतश्यावानामर्चिष्मतां च रूपाणां स्वप्ने दर्शनमभीक्ष्णमिति (लोहितपित्तपूर्वरूपाणि भवन्ति)॥ *C.S.Ni. 2/6*

सदनं शीतकामित्वं कण्ठधूमायनं वमिः ।
लोहगन्धिश्च निःश्वासो भवत्यस्मिन् भविष्यति ॥

S.S.Utt. 45/8

Premonitory signs and symptoms of the disease *Raktapitta* are as follows : Lack of desire to take food, burning sensation in *Mahāsrotas* due to improper digestion, eructations having sour taste and smell like vinegar, frequent urge for vomiting, foul smell from vomited material, hoarseness of voice, general malaise, burning sensation all over the body, patients feels as if smoke is emitting from oral cavity, smell like iron metal, fish and raw flesh from mouth; red, green and yellow discolouration in different body parts, stool, urine, sweat, saliva and excreta from nose, mouth, ear and eyes; appearance of pimples; bodyache; visualizes red, blue, yellow, and brown colour objects and dazzling objects in dreams, desire for cold things; patients feels as if fumes are coming out from throat; expiratory smell like iron metal.

रूप (Rupa)

Signs and symptoms as per the involvement of pathways and their prognosis :

मार्गौ पुनरस्य द्वौ-ऊर्ध्वं, चाधश्च। तद्बहुश्लेष्मणि शरीरे श्लेष्मसंसर्गादूर्ध्वं प्रतिपद्यमानं कर्णनासिकानेत्रास्येभ्यः प्रच्यवते, बहुवाते तु शरीरे वातसंसर्गादधः प्रतिपद्यमानं मूत्रपूरीषमार्गाभ्यां प्रच्यवते, बहुश्लेष्मवात तु शरीरे श्लेष्मवातसंसर्गाद्द्वावपि मार्गौ प्रतिपद्यते, तौ मार्गौ प्रतिपद्यमानं सर्वेभ्य एव यथोक्तेभ्यः खेभ्यः प्रच्यवते शरीरस्य॥

तत्र यदूर्ध्वभागं तत् साध्यं, विरेचनोपक्रमणीयत्वाद्बह्वौषधत्वाच्च; यदधोभागं तद्याप्यं, वमनोपक्रमणीयत्वादल्पौषधत्वाच्च; यदुभयभागं तदसाध्यं, वमनविरेचनायोगित्वादनौषधवाच्चेति॥

C.S.Ni. 2/8-9

Disease *Raktapitta* takes its root either upwards or downwards.

ऊर्ध्वग रक्तपित्त (Ūrdvaga Raktapitta)

In case of *Ūrdvaga Raktapitta, Kapha Doṣa* dominates and patients expels the blood from ear, nose, eyes, and mouth.

It is curable because of its amenability to purgation therapy and also lot of drugs are available to treat this condition.

अधोग रक्तपित्त (Adhoga Raktapitta)

It manifests due to predominance of *Vāta* and patients expel the blood from lower tracks i:e urethra and anus.

It is mangeable because of its amenability to emetic therapy and availability of drugs are limited.

उभय रक्तपित्त/द्वावपि मार्ग (Ubhaya Raktapitta/ Dvāvapi Mārga)

It results due to predominance of *Vāta* and *Kapha* and patient expels the blood from both upper and lower tracks.

It is incurable because it is neither amenable to purgation nor to emetic therapy and non availability of suitable drugs.

विशेष लक्षण (Viśeṣa Lakṣaṇa)

सान्द्रं सपाण्डु सस्नेहं पिच्छिलं च कफान्वितम् ।
श्यावारुणं सफेनं च तनु रूक्षं च वातिकम् ॥

C.S.Ci. 4/11

बाह्यासृग्लक्षणैस्तस्य सङ्ख्यादोषोच्छ्रतीविदुः ॥

S.S.Utt. 45/8

तत्र, फेनिलमरुणं कृष्णं परुषंतनु शीघ्रगमस्कन्दि च वातेन दुष्टम । नीलं पीतं हरितं, श्यावं विस्त्रमनिष्टं पिपीलिकामक्षिकाणामस्कन्दि च पित्तदुष्टम् । गैरिकोदकप्रतीकाशं स्निग्धं शीतलं बहलं पिच्छिलं चिरस्त्रावि मांसपेशीप्रभं च श्लेष्मदुष्टम् । सर्वलक्षणसंयुक्तं काञ्जिकाभं विशेषतो दुर्गन्धि च सन्निपातदुष्टं, पित्तवद्रक्तेनातिकृष्णं च द्विदोषलिङ्गं संसृष्टम् । *S.S.Su. 14/21*

वातदुष्टि रक्त लक्षण/वातज रक्तपित्त (Vātaduṣṭi Rakta Lakṣaṇa/ Vātaja Raktapitta Lakṣaṇa)

Symptoms of vitiated *Rakta* due to *Vāta* are as follows : Blood becomes froathy, blackish, reddish, thin, ununctous, nonslimy, flowing quickly and not clotting.

पित्तदुष्टि रक्त लक्षण/पित्तज रक्तपित्त लक्षण (Pittaduṣṭi Rakta Lakṣaṇa/Pittaja Rakta Pitta Lakṣaṇa)

Symptoms of vitiated *Rakta* due to *Pitta* are as follows : Blood becomes ochre like (pink red), black, appear like cow's urine, shining black, smoky (house-shoot), black collyrium, bluish yellow, green or black, unpleasant smell; flies and ants are not attracted and doesn't clot.

कफदुष्टि रक्त लक्षण/कफज रक्तपित्त लक्षण (Kapha-duṣṭi Rakta Lakṣaṇa/Kaphaja Raktapitta Lakṣaṇa)

Symptoms of vitiated *Rakta* due to *Kapha* are as follows : Blood becomes dense, pale yellow, unctuous, slimy, appear like red ochre (*Gairika*), cold, thick, flowing slowly and resemble like muscle tissue.

द्विदोषलिङ्ग लक्षण (Dvidoṣa Liṅga Lakṣaṇa) Or द्वन्द्वज रक्तपित्त लक्षण (Dvandvaja Raktapitta Lakṣaṇa)

Symptoms of vitiated blood due to two *Doṣa* involvement are as follows:- Features of any two *Doṣās* will be observed.

त्रिदोषज दुष्टि रक्त लक्षण (Tridoṣaja Duṣṭi Rakta Lakṣaṇa) Or सन्निपातज रक्तपित्त (Sannipātaja Raktapitta)

Symptoms of vitiated blood due to all the three *Doṣās* are as follows : Symptoms of all the three *Doṣās* resemble like rice gruel and having bad smell.

उपद्रव (Upadrava)

दौर्बल्यश्वासकासज्वरवमथुमदास्तन्द्रितादाहमूर्च्छा भुक्ते चान्ने विदाहस्त्वधृतिरपि सदा हृद्यतुल्या च पीडा ।

तृष्णा कण्ठस्य भेदः शिरसि च दवनं पूतिनिष्ठीवनञ्च द्वेषो भक्तेऽविपाको विरतिरपि रते रक्तपित्तोपसर्गाः ॥

S.S.Utt. 45/10

उपद्रवास्तु खलु दौर्बल्यारोचकाविपाकश्वासकास-ज्वरातीसारशोफशोषपाण्डुरोगाः स्वरभेदश्च॥ *C.S.Ni. 2/7*

Complications of *Raktapitta* are as follows : Debility, dyspnoea, cough, fever, vomiting, toxaemia, stupor, burning sensation, fainting, burning sensation after taking food, anxiety, constant pain in cardiac region, thirst, abnormal voice, severe pain in head, bad smell from sputum, aversion to food, indigestion, lack of sexual desire, fever, diarrhoea, oedema, consumption and anaemia.

साध्यासाध्यता (Sādhyāsādhyatā)

एकदोषानुगं साध्यं द्विदोषं याप्यमुच्यते ॥
यत्रिदोषमसाध्यं तन्मन्दाग्नेरतिवेगवत् ।
व्याधिभिः क्षीणदेहस्य वृद्धस्यानश्नतश्च यत् ॥

C.S.Ci. 4/13-14

Raktapitta is curable if it associates with single *Doṣa*, it is palliable if it associates with two *Doṣās*; it is incurable if it associates with all the three *Doṣās*. Patients of *Raktipitta* becomes incurable if patients suffers from *Mandāgni*, having an episode of acute attacks, emaciation, old and one who is not able to eat.

यदा तु सर्वच्छिद्रेभ्यो रोमकूपेभ्य एव च ।
वर्तते तामसङ्ख्येयां गतिं तस्याहुरान्तिकीम् ॥
यच्चोभयाभ्यां मार्गाभ्यामतिमात्रं प्रवर्तते ।
तुल्यं कुणपगन्धेन रक्तं कृष्णमतीव च ॥
संसृष्टं कफवाताभ्यां कण्ठे सज्जति चापि यत् ।
यच्चाप्युपद्रवैः सर्वैर्यथोक्तैः समभिद्रुतम् ॥
हारिद्रनीलहरितताम्रैर्वर्णैरुपद्रुतम् ।
क्षीणस्य कासमानस्य यच्च तच्च न सिध्यति ॥
यद्द्विदोषानुगं यद्वा शान्तं प्रकुप्यति ।
मार्गान्मार्गं चरेद्यद्वा याप्यं पित्तमसृक् च तत् ॥

C.S.Ci. 4/17-21

If blood moves through all the holes in the roots of hairs which are innumerable and this indicates death.

Disease becomes incurable in the following conditions : Excess bleeding from either of the tracts; smell becomes like dead body; colour changed to extremely black; association with both *Vāta* and *Kapha;* if blood gets obstructed in the throat; presence of all the *Upadrava;* colour of blood changed to deep yellow, blue, green or coppery associated with emaciation and coughing.

Rakta pitta becomes manageable in the following conditions

Association of two *Doṣās* & two tracks; recurrent episodes of attacks and shifts from one passage to another.

एकमार्गं बलवतो नातिवेगं नवोत्थितम् ।
रक्तपित्तं सुखे काले साध्यं स्यान्निरुपद्रवम् ॥

(C.S.Ci. 4/22)

Rakta Pitta is curable in the following patients

- Only one track is involved;
- Physically strong; mild attacks; recent onset; favourable environment for management and patient is free from complications.

अरिष्ठ लक्षण (Ariṣṭha Lakṣaṇa)

मांसप्रक्षालनाभं क्वथितमिव च यत् कर्दमाम्भोनिभं वा
मेदःपूयास्रकल्पं यकृदिव यदि वा पक्वजम्बूफलाभम्। यत् कृष्णं
यच्च नीलं भृशमतिकुणपं यत्र चोक्ता विकारास्तद्वर्ज्यं रक्तपित्तं
सुरपतिधनुषा यच्च तुल्यं विभाति ॥ *S.S.Utt. 45/11*

Expelled blood resembles like mutton wash, decoction or muddy water; it may mixed with fat or pus and resemble like liver or ripened Jambu phala; blood is either black or blue and emits cadaveric smell; shines like the rainbow and associated with complications should be rejected for treatment.

उपशय/पथ्य (Upaśaya/Pathya) C.S.Ci. 4th Chapter

अन्न (Anna) S.S.Utt. 45th Chapter

Laṅgana, Śāli, Ṣaṣṭika, Nīvāra, Kora dūṣa, Mudga, Masūra, Caṇaka, Paṭola, Nimba, Plakṣa, Kirātatikta, Kaṭhillaka.

औषध (Auṣadha)

Virecana, Vāsāghṛta, Śatāvaryādi ghṛta etc.

The following items of food are useful in cases of *Raktapitta*: old rice, barley, *Mudga* (phaseolus bean), lentil, gram, pigeon pea and kidney bean, ŕŕraw sugar, honey, sugarcane, *Ghṛtam*, banana, jackfruit, almond, *Paṭola*, shoot of willow, *Kirātatikta*, stem of *Kamala*, leaves of *Neem*, pomegranate, coconut, wood, apple and raisin, The patient can take meat of a number of animals and birds including rabit, deers, pigeon, swans, ducks, partridge, wood-cutter and others. Moreover, *Virecana*, massage, plaster of the sandlewood paste on the body and bathing, etc. are good for the patients. (B.R. 13/167-176)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Following is the list which should be avoided by a person suffering from *Rakta Pitta* disorders: wrestling (or similar physical exercises), walking, roaming under direct sun, doing evil deeds, depression, disturbing the flow of excretions, instablity (getting up frequently), horse and elephant riding, blood letting, *Svedana Karma*, smoking, sex, anger, *Kulattha*, jaggery, brinjal, sesame, *Urada*, mustard, yogurt, water from well, betel, water treated with *Uśīra*. The prescription of avoidable also includes, *Āmla*, salty and bitter juices, seeds of *Kulattha* (dolichos bean) and sesame, legumes, leaf of mustard, brinjal and garlic pearls, *Sema*, consumption of incomptible, *Kaṭu*, *Āmla* and citrus products, consumption of salty products and products which produces burning sensation. (B.R. 13/177-179).

MCQs

1. *Charditasya bibhatsata* is the feature of
   a. *Raktapitta Purvarupa*
   b. *Raktapittarupa*
   c. *RaktapittaAsadyalakshana*
   d. *Raktapittaupadrava*
2. According to *Charaka*, which one of the statements about the prognosis of *Raktapitta* is correct
   a. It is curable in individuals who are physically weak with moderate attacks and chronic onset with favorable environment for management associated with complications
   b. It is curable in individuals who are physically weak with severe attacks and recent onset with no favorable environment for management associated with complications
   c. It is curable in individuals who are physically strong with mild attacks and recent onset with favorable environment for management associated with free from complications
   d. It is curable in individuals who are physically strong with mild attacks and recent onset with favorable environment for management associated with complications
3. Number of *Rakthapitta Upadrava* as per *Madavanidana*
   a. 7 b. 8
   c. 12 d. 18

4. Which among the following is not the *Asadyalakshana* of *Raktapitta*
    - a. *Mamsaprakshalanabham*
    - b. *Pakwajambhuphalabham*
    - c. *Gomutrasannibha*
    - d. *Brisamatikunapa*
5. Prognosis of *Adhoga Raktapitta*
    - a. *Sadyam*
    - b. *Krichrasadya*
    - c. *Yapya*
    - d. *Asadhya*
6. *Dhoomako-amlaka is*
    - a. *Purvarupa of Raktapitta*
    - b. *Pitta kasa lakshana*
    - c. *Upadrava of Kasa*
    - d. *Both a & b*
7. *Bhukteghoravidaha* is the *Upadrava* of
    - a. *Raktapitta*
    - b. *Pandu*
    - c. *Chardhi*
    - d. *Ajeerna*

Answer

1.	a	2.	c	3.	d
4.	c	5.	c	6.	d
7.	a				

CHAPTER

22

Hematopoietic diseases

Anaemia

1. Anaemia refers to a state in which the level of haemoglobin in the blood is below the normal range appropriate for age and sex.
2. Other factors, including pregnancy and altitude, also affect haemoglobin levels and must be taken into account when considering whether in individual is anaemic.
3. The clinical features of anaemia reflect diminished oxygen supply to the tissues
4. A rapid onset of anaemia (e.g. due to blood loss) causes more profound symptoms than a gradually developing anaemia.
5. Individual with cardio respiratory diseases are more susceptible to symptoms of anaemia.
6. Around 30 % of the total world population is anaemic and half of these, some 600 million people, have iron deficiency.
7. The classification of anaemia by the size of the red cells (MCV) indicates the likely cause.
8. Red cells in the bone marrow must acquire a minimum level of haemoglobin before being released into the blood stream.
9. Whilst in the marrow compartment, red cell precursors undergo cell division driven by erythropoietin.
10. If red cells cannot acquire haemoglobin at a normal rate, they will undergo more divisions than normal and will have a low MCV when finally released into the blood.
11. The MCV is low because component parts of the haemoglobin molecule are not fully available: that is, iron in iron deficiency, globin chains in thalassaemia, haem ring in congenital sideroblastic anaemia and, occasionally, poor iron utilisation in the anaemia of chronic disease.
12. In megaloblastic anaemia the biochemical consequence of vitamin B_{12} or folate deficiency is an inability to synthesise new bases to make DNA.
13. A similar defect of cell division is seen in the presence of cytotoxic drugs or haematological disease in the marrow such as myelodysplasia.
14. In these states, cells haemoglobinise normally but undergo fewer cell divisions, resulting in circulating red cells with a raised MCV.
15. The red cell membrane is composed of a lipid bilayer which will freely exchange with the plasma pool of lipid.
16. Conditions such as liver disease, hypothyroidism, hyperlipidaemia and pregnancy are associated with raised lipids and may also cause a raised MCV
17. Reticulocytes are larger than mature red cells, so when the reticulocyte count is raised-for example, in haemolysis-this may also increase the MCV.

Clinical assessment

1. Iron deficiency anaemia is the most common type of anaemia world-wide
2. A thorough gastrointestinal history is important, looking in particular for symptoms of blood loss.
3. Menorrhagia is a common cause of anaemia in pre-menopausal females, so women should always be asked about their periods.
4. A dietary history should assess the intake of iron and folate which may become deficient in comparison to needs (e.g. in pregnancy or during periods of rapid growth.

Past medical history

Diseases known to be associated with anaemia,

- Rheumatoid arthritis (the anaemia of chronic disease)
- Previous surgery (e.g. resection of the stomach

or small bowel which may lead to malabsorption of iron and/or vitamin B_{12}).

Family history and ethnic background

- It may raise suspicion of haemolytic anaemias such as the haemoglobinopathies and hereditary spherocytosis.
- Pernicious anaemia may also be familial.

A drug history : It may reveal the ingestion of drugs which cause blood loss (e.g. aspirin and antiinflammatory drugs), haemolysis or aplasia.

Investigations

1. Schemes for the investigation of anaemias are often based on the size of the red cells, which is most accurately indicated by the MCV in the FBC. Commonly the presence of anaemia.
2. A normal MCV (normocytic anaemia) suggests either acute blood loss or the anaemia of chronic disease (ACD)
3. A low MCV (microcytic anaemia) suggests iron deficiency or thalassaemia
4. A high MCV (macrocytic anaemia) suggests vitamin B_{12} or folate deficiency or myelodysplasia.

Classification of Anaemias

Several types of classifications of anaemias have been proposed. Two of the widely accepted classifications are based on the pathophysiology and morphology

Pathophysiologic Classification

Depending upon the pathophysiologic mechanism, anaemias are classified into 3 groups:

I. Anaemia due to blood loss -This is further subdivided into 2 types:

A. Acute post-haemorrhagic anaemia

B. Anaemia of chronic blood loss

II. Anaemia due to impaired red cell formation -A disturbance due to impaired red cell production from various causes may produce anaemia. These are as under :

A. Cytoplasmic maturation defects

1. Deficient haem synthesis : iron deficiency anaemia
2. Deficient globin synthesis : thalassaemic syndromes

B. Nuclear maturation defects

Vitamin B_{12} and/or folic acid deficiency: megaloblastic anaemia

C. Haematopoietic stem cell proliferation and differentiation abnormality e.g.

1. Aplastic anaemia
2. Pure red cell aplasia

D. Bone marrow failure due to systemic diseases (anaemia of chronic disorders) e.g.

1. Anaemia of inflammation/infections, disseminated malignancy
2. Anaemia in renal disease
3. Anamia due to endocrine and nutritional deficiencies (hypometabolic states)
4. Anaemia in liver disease

E. Bone marrow infiltration e.g.

1. Leukaemias
2. Lymphomas
3. Myelosclerosis
4. Multiple myeloma

F. Congenital anaemia e.g.

1. Sideroblastic anaemia
2. Congenital dyserythropoietic anaemia.

The term hypoproliferative anaemias is also used to denote impaired marrow proliferative activity and includes 2 main groups : hypoproliferation due to iron deficiency and that due to other hypoproliferative disorders; the latter category includes anaemia of chronic inflammation/infection, renal disease, hypometabolic states, and causes of bone marrow failure.

III. Anaemia due to increased red cell destruction (haemolytic anaemias) This if further divided into 2 groups :

A. Intracorpuscular defect (hereditary and acquired haemolytic anaemias).

Morphologic Classification

Based on the red cell size, haemoglobin content and red cell indices, anaemias are classified into 3 types:

1. **Microcytic, hypochromic** - MCV, MCH, MCHC are all reduced e.g. in iron deficiency anaemia and in certain non-iron deficient anaemias (sideroblastic anaemia, thalassaemia, anaemia of chronic disorders).
2. **Normocytic, normochromic**- MCV, MCH, MCHV are all normal e.g. after acute blood loss, haemolytic anaemias, bone marrow failure, anaemia of chronic disorders.
3. **Macrocytic** -MCV is raised e.g. in megaloblastic anaemia due to deficiency of vitamin B_{12} or folic acid.

High yield information

- * Anaemia is reduced haemoglobin concentration in blood below the lower limit of the normal for the age and sex of the individual.
- * Anaemia causes lowered oxygen-carrying capacity and eventually tissue hypoxia and impaired function.
- * Major symptoms of anaemia are tiredness, weakness and lethargy and main signs are pallor, hyperdynamic circulation, and impaired function of CNS and kidney.
- * A suspected case of anaemia is investigated by haemoglobin estimation, peripheral blood smear examination, and complete blood counts including reticulocyte count and ESR.
- * Bone marrow aspiration and trephine biopsy are done to confirm and type the anaemia.
- * Anaemias are classified based on pathophysiology (into anaemia due to blood loss, impaired red cell destruction) or morphology (into microcytic hypochromic, macrocytic, normocytic normochromic).

Classification of anaemias in brief

A. Pathophysioloigic
I. Anaemia due to increased blood loss
  a) Acute post-haemorrhagic anaemia
  b) Chronic blood loss
II. Anaemias due to impaired red cell production
  a) Cytoplasmic maturation defects
    1. Deficient haem synthesis : Iron deficiency anaemia
    2. Deficient globin synthesis :
Thalassaemic syndromes
  b) Nuclear maturation defects
    Vitamin B_{12} and /or folic acid deficiency : Megaloblastic anaemia
  c) Defect in stem cell proliferation and differentiation
    1. Aplastic anaemia
    2. Pure red cell aplasia
  d) Anaemia of chronic disorders
  e) Bone marrow infiltration
  f) Congenital anaemia
III. Anaemias due to increased red cell destruction (Haemolytic anaemias)
A. Extrinsic (extracorpuscular) red cell abnormalities
B. Intrinsic (intracorpuscular) red cell abnormalities
C. Morphologic
I. Microcytic, hypochromic
II. Normocytic, Normochromic
III. Macrocytic, normochromic

Iron deficiency anaemia

It occurs when iron losses or physiological requirements exceed absorption.

Aetiology

A. Blood loss :

1. The most common explanation in men and postmenopausal women is gastrointestinal blood loss
2. In developed countries one-third of premenopausal women have low iron stores but only 3% display iron-deficient haematopoiesis.
3. Very rarely, chronic haemoptysis or haematuria may cause iron deficiency.

B. Malabsorption :

1. Gastric acid is required to release iron from food and helps to keep iron in the soluble ferrous state
2. Hypochlorhydria in the elderly or that due to

drugs such as proton pump inhibitors may contribute to the lack of iron availability from the diet, as may previous gastric surgery.

3. Iron is absorbed actively in the upper small intestine and hence can be affected by coeliac disease

C. Physiological demands

1. At times of rapid growth, such as infancy and puberty, iron demands increase and may outstrip absorption.

Investigation of the cause

1. It depend upon the age and sex of the patient, as well as the history and clinical findings.
2. The upper and lower gastrointestinal tract should be investigated by endoscopy or barium studies
3. Serum antiendomysial antibodies and possibly a duodenal biopsy are indicated to detect coeliac disease in irondeficient patients who have features of malabsorption or recurrent deficiency in the absence of other explanations, or who are young men with normal diet or young women with normal menstruation and diet.
4. In the tropics, stool and urine should be examined for parasites
5. *Confirmation of iron deficiency plasma* ferritin is a measure of iron stores in tissues and is the best single test to confirm iron deficiency.
6. Plasma iron and total iron binding capacity (TIBC) are measures of iron availability
7. Levels of transferrin, the binding protein for iron

Anaemia of chronic disease (ACD)

1. This is a common type of anaemia, particularly in hospital populations.
2. It occurs in the setting of chronic infection, chronic inflammation or neoplasia.
3. The anaemia is not related to bleeding, haemolysis or marrow infiltration, is mild, with Hb in the range of 85-115g/ L, and is usually associated with a normal MCV (normocytic, normochromic), though this may be reduced in long-standing inflammation.
4. The serum iron is low but iron stores are normal or increased, as indicated by the ferritin or stainable marrow iron.

Pathogenesis

1. It has recently become clear that the key regulatory protein that accounts for the findings characteristic of ACD is hepcidin, which is produced by the liver.
2. High levels of production are encouraged by proinflammatory cytokines, especially IL-6. Hepcidin binds to ferroportin on the membrane of iron exporting cells, such as small intestinal enterocytes and macrophages, internalising the ferroportin and thereby inhibiting the export of iron from these cells into the blood (and hence to the main target cells and proteins of iron).
3. The iron remains trapped inside the cells in the form of ferritin, levels of which are therefore normal or high in the face of significant anaemia.
4. Inhibition or blockade of hepcidin is a likely target for potential treatment of this form of anaemia.

Megaloblastic anaemia

Aetiology : This results from a deficiency of vitamin B_{12}, or folic acid or from disturbances in folic acid metabolism.

Pathogenesis

Deficiency of either vitamin B_{12}, or folate

↓

Produce high plasma levels of homocysteine and impaired DNA synthesis

↓

The end result is cells with arrested nuclear maturation but normal cytoplasmic development: so-called nucleocytoplasmic asynchrony.

↓

All proliferating cells will exhibit megaloblastosis

↓

Evident changes observed in the buccal mucosa, tongue, small intestine, cervix, vagina and uterus.

↓

Megaloblastic Anaemia

Dietary deficiency

This only occurs in strict vegans but the onset of clinical features can occur at any age between 10 and 80 years.

Gastric factors

Release of vitamin B_{12} from the food requires normal gastric acid and enzyme secretion, and this is impaired by hypochlorhydria in elderly patients or following gastric surgery.

Pernicious anaemia

This is an autoimmune disorder in which the gastric mucosa is atrophic, with loss of parietal cells causing intrinsic factor deficiency.

Small bowel factors

One-third of patients with pancreatic exocrine insufficiency fail to transfer dietary vitamin B_{12} from R protein to intrinsic factor.

Clinical features of megaloblastic anaemia

1. Malaise
2. Impotence
3. Breathlessness
4. Poor memory
5. Paraesthesiae
6. Depression
7. Sore mouth
8. Personality change
9. Weight loss
10. Hallucinations
11. Altered skin pigmentation
12. Visual disturbance
13. Grey hair

Signs

1. Smooth tongue
2. Skin pigmentation
3. Angular cheilosis
4. Heart failure
5. Vitiligo
6. Pyrexia

Investigations in megaloblastic anaemia

1. Haemoglobin-Often reduced, may be very low
2. MCV-usually raised, commonly > 120 fL
3. Erythrocyte count-Low for degree of anaemia
4. Blood film-Oval macrocytosis, poikilocytosis, red cell fragmentation, neutrophil hypersegmentation
5. Reticulocyte count-Low for degree of anaemia
6. Leucocyte count-Low or normal
7. Platelet count- Low or normal
8. Bone marrow Increased cellularity, megaloblastic changes in erythroid series, giant metamyelocytes, dysplastic megakaryocytes, increased iron in stores, pathological non-ring sideroblasts
9. Serum ferritin - Elevated
10. Plasma lactate - Elevated, often markedly

Folate deficiency

1. Folates are produced by plants and bacteria; hence dietary leafy vegetables (spinach, broccoli, lettuce), fruits (bananas, melons) and animal protein (liver, kidney) are a rich source.
2. An average Western diet contains more than the minimum daily intake of 50gm.
3. Excess cooking for longer than 15 minutes destroys folates.

Causes of folate deficiency

Diet—Poor intake of vegetables

Malabsorption—e.g. Coeliac disease

Increased demand-Cell proliferation, e.g. Hamolysis; Pregnancy

Drugs—Certain anticonvulsants (e.g. phenytoin); certain cytotoxic drugs (e.g. methotrexate)

Investigation to diagnose folic acid deficiency

1. Low serum folate levels (fasting blood sample)
2. Red cell folate levels low (but may be normal if folate deficiency is of very recent onset)
3. Macrocytic dysplastic blood picture
4. Megaloblastic marrow

Haemolytic anaemia

1. The normal red cell lifespan of 120 days may be shortened by a variety of abnormalities.
2. The bone marrow may increase its output of red cells six- to eight-fold by increasing the proportion of red cells produced, expanding the volume of active marrow and releasing reticulocytes prematurely.
3. If the rate of destruction exceeds this increased production rate, then anaemia will develop.
4. Red cell destruction overloads pathways for haemoglobin breakdown, causing a modest rise in unconjugated bilirubin in the blood and mild jaundice.

5. Increased reabsorption of urobilinogen from the gut results in an increase in urinary urobilinogen.
6. Activation of the bone marrow can result in a neutrophilia and immature granulocytes appearing in the blood to cause a leuco-erythroblastic blood film.
7. The appearance of the red cells may give an indication of the likely cause of the hemolysis.
8. Spherocytes are small, dark red cell which suggests autoimmune homolysis or hereditary spherocytosis.
9. Sickle cells suggest hemoglobinopathy
10. Red cell fragments indicate microangiopathic haemolysis
11. The compensatory erythroid hyperplasia may give rise to folate deficiency, when the blood findings will be complicated by the presence of megaloblastosis.
12. Measuement of red cell folate is unreliable in the presence of haemolysis and serum folate will be elevated.

Anemias

Classification of anemias according to morphology :

1. Hypochromic microcytic anemias
   a. Iron deficiency anemia
   b. Thalassemias, hemoglobinopathies
   c. Sideroblastic anemia
   d. Anemia of chronic disorders
   e. Lead poisoning
   f. Vitamin B_6 deficiency
2. Normochromic normocytic anemias
   a. Anemia due to acute blood loss
   b. Hemolytic anemias
   c. Anemia due to bone marrow failure, renal disease, etc.
   d. Anemia of chronic disorders
3. Macrocytic anemias
   a. Megaloblastic anemia : Vitamin B_{12} or folate deficiency.
   b. Non-megaloblastic anemias : Liver disease, myelodysplasia, aplastic anemia, drug-induced anemia, hemolysis, hypothyroidism, alcoholism, etc.

Nutritional Anemias

Anemia is defined as a reduction below normal in the concentration of hemoglobin (Hb) or red blood cells in the blood. In India, hemoglobin levels below 13 g/dl in males, 12 g/dl in non-pregnant females, and 11 g/dl in pregnant females should be diagnosed as anemia.

Anemias may be classified based on red cell morphology as given below :

1. Microcytic hypochromic : Iron deficiency, thalassaemias Chronic infection, sideroblastic anemia
2. Normocytic normochromic : Aplastic anaemia, post-haemorrhagic
3. Macrocytic normochromic : Folate deficiency, vitamin B_{12} deficiency
4. Macrocytic hypochromic : Combined deficiency of iron and folate or B_{12}

The specific management depends on the etiological diagnosis described in the relevant chapters in this book and other text books of kayachikitsa vol I & II.

Thalassaemia

1. Thalassaemia is an inherited impairment of haemoglobin production, in which there is partial or complete failure to synthesise a specific type of globin chain.
2. In alpha-thalassaemia, disruption of one or both alleles on chromosome 16 may occur, with production of some or no alpha globin chains.
3. In beta-thalassaemia, defective production usually results from disabling point mutations causing no (3^0) or reduced (3-) beta chain production.

Beta-thalassaemia

Failure to synthesise beta chains (beta-thalassaemia) is the most common type of thalassaemia, most prevalent in the Mediterranean area. Heterozygotes have thalassaemia minor, a condition in which there is usually mild anaemia and little or no clinical disability, which may be detected only when iron therapy for a mild microcytic anaemia fails. Homozygotes (thalassaemia major) either are unable to synthesise haemoglobin A or at best produce very little; after the first 4-6 months of life they develop profound hypochromic anaemia.

Sickle-cell anaemia

1. It results from a single glutamic acid to valine substitution at position 6 of the beta globin polypeptide chain.
2. It is inherited as an autosomal recessive trait

Pathogenesis

When haemoglobin S is deoxygenated

↓

The molecules of haemoglobin polymerise to form pseudocrystalline structures known as 'tactoids'.

↓

These distort the red cell membrane

↓

Produce characteristic sickle-shaped cells

Clinical features

1. Hypoxia
2. Acidosis
3. Dehydration
4. Infection.
5. Irreversibly sickled cells have a shortened survival and plug vessels in the microcirculation.
6. This results in a number of acute syndromes, termed 'crises', and chronic organ damage

Investigations

- Compensated anaemia, usually around 60-80g/h.
- The blood film shows sickle cells, target cells and features of hyposplenism.
- A reticulocytosis is present.
- The presence of HbS can be demonstrated by exposing red cells to a reducing agent such as sodium dithionite; HbA gives a clear solution, whereas HbS polymerises to produce a turbid solution.
- The definitive diagnosis requires haemoglobin electrophoresis to demonstrate the absence of HbA, 2-20% HbF and the predominance of HbS.
- Both parents of the affected individual will have sickle-cell trait.

Leukaemias

1. Leukaemias are malignant disorders of the haematopoietic stem cell compartment, characteristically associated with increased numbers of white cells in the bone marrow and/or peripheral blood.
2. The course of leukaemia may vary from a few days or weeks to many years, depending on the type.

Epidemiology

The incidence of leukaemia of all types in the population is approximately 10/100 000 per annum, of which just under half are cases of acute leukaemia.

1. Males are affected more frequently than females
   - 3:2 in acute leukaemias
   - 2:1 in chronic lymphocytic leukaemia
   - 1.3:1 in chronic myeloid leukaemia.
2. Geographical variation in incidence does occur, the most striking being the rarity of chronic lymphocytic leukaemia in the Chinese and related races.
3. Acute leukaemia occurs at all ages.
4. Acute lymphoblastic leukaemia shows a peak of incidence in children aged 1-5 years.
5. All forms of acute myeloid leukaemia have their lowest incidence in young adult life and there is a striking rise over the age of 50.
6. Chronic leukaemias occur mainly in middle and old age.

Aetiology

1. The cause of the leukaemia is unknown in the majority of patients.
2. Risk factors for leukaemia

Ionising radiation

- After atomic bombing of Japanese cities (myeloid leukaemia) ,
- Radiotherapy for ankylosing spondylitis
- Diagnostic X-rays of the fetus in pregnancy

Cytotoxic drugs

- Especially alkylating agents (myeloid leukaemia, usually after a latent period of several years)
- Industrial exposure to benzene

Retroviruses

One rare form of T-cell leukaemia/lymphoma appears to be associated with a retrovirus similar to the viruses causing leukaemia in cats and cattle

Genetic

- Identical twin of patients with leukaemia
- Down's syndrome and certain other genetic disorders

Immunological

Immune deficiency states (e.g. hypogammaglobulinaemia)

Terminology and classification

Leukaemias are traditionally classified into four main groups:

- Acute lymphoblastic leukaemia (ALL)
- Acute myeloid leukaemia (AML)
- Chronic lymphocytic leukaemia (CL.L)
- Chronic myeloid leukaemia (CML).

In acute leukaemia there is proliferation of primitive stem cells leading to an accumulation of blasts, predominantly in the bone marrow, which causes bone marrow failure.

↓

In chronic leukaemia the malignant clone is able to differentiate, resulting in an accumulation of more mature cells.

↓

Lymphocytic and lymphoblastic cells are those derived from the lymphoid stem cell (B cells and T cells).

↓

Myeloid refers to the other lineages, i.e. precursors of red cells, granulocytes, monocytes and platelets.

Diagnosis of leukaemia

1. Blood test

   Raised white count

2. Confirmed by examination of the bone marrow.

   This includes the morphology of the abnormal cells, analysis of cell surface markers (immunophenotyping), clone-specific chromosome abnormalities and molecular changes.

3. The features in the bone marrow not only provide an accurate diagnosis but also give valuable prognostic information, allowing therapy to be tailored to the patient's disease.

Acute leukaemia

1. There is a failure of cell maturation in acute leukaemia.
2. Proliferation of cells which do not mature leads to an accumulation of useless cells which take up more and more marrow space at the expense of the normal haematopoietic elements.
3. Eventually this proliferation spills into the blood.
4. Acute myeloid leukaemia (AML) is about four times more common than acute lymphoblastic leukaemia (ALL) in adults.
5. In children the proportions are reversed, the lymphoblastic variety being more common.

The clinical features

Bone marrow failure features

1. Anaemia 2. Bleeding 3. Infection

Investigations

1. Blood examination
   - Anaemia with a normal or raised MCV
   - The leukocyte count may vary from as low as 1 x 10^9/L to as high as 500 x 109/L or more.
   - In the majority of patients the count is below 100 x 10^9/ L.
   - Severe thrombocytopenia is usual but not invariable.
   - The appearance of blast cells in the blood film is usually diagnostic.
   - Sometimes the blast cell count may be very low in the peripheral blood and a bone marrow examination is necessary to confirm the diagnosis.

2. The bone marrow : Hypercellular, with replacement of normal elements by leukaemic blast cells in varying degrees of the cells. The presence of Auer rods in the cytoplasm of blast cells indicates a myeloblastic type of leukaemia.

Chronic myeloid leukaemia (CML)

1. It is a myeloproliferative stem cell disorder resulting in proliferation of all haematopoietic lineages but manifesting predominantly in the granulocytic series.
2. Maturation of cells proceeds fairly normally.
3. The disease occurs chiefly between the ages of 30 and 80 years, with a peak incidence at 55 years.
4. It is rare and accounts for 20% of all leukaemias.
5. The disease is found in all races.
6. Approximately 95% of patients with CML have a chromosome abnormality known as the Philadelphia (Ph) chromosome.

Natural history

The disease has three phases:

1. A chronic phase
   Which the disease is responsive to treatment and is easily controlled, typically lasting 3-5 years
2. An accelerated phase (not always seen), in which disease control becomes more difficult.
3. *Blast crisis,* in which the disease transforms into an acute leukaemia, either myeloid (70%) or lymphoblastic (30%), which is relatively refractory to treatment. This is the cause of death in the majority of patients; therefore survival is dictated by the timing of blast crisis, which cannot be predicted.

Clinical features of chronic myeloid leukaemia

1. Tiredness
2. Weight lass
3. Breathlessness
4. Abdominal pain and discomfort
5. Lethargy
6. Anorexia
7. Sweating
8. Abdominal fullness
9. Bruising
10. Vague ill health

Physical examination

1. Splenomegaly is present in 90%; in about 10% the enlargement is massive, extending to over 15cm below the costal margin.
2. A friction rub may be heard in cases of splenic infarction.
3. Hepatomegaly occurs in about 50% Lymphadenopathy is unusual.

Investigations

1. Full blood count (FBC)
   - A normocytic, normochromic anaemia
   - The leucocyte count can vary from 10 to 600 x 10^9/ L.
   - In about one-third of patients there is a very high platelet count, sometimes as high as 2000 x 10^9/L.
   - In the blood film the full range of granulocyte precursors from myeloblasts to mature neutrophils is seen but the predominant cells are neutrophils and myelocytes
   - Myeloblasts usually constitute less than 10% of all white cells.
   - There is often an absolute increase in eosinophils and basophils
   - Nucleated red cells are common.
2. Bone marrow
   - It should be obtained to confirm the diagnosis and phase of disease by morphology
   - Chromosome analysis to demonstrate the presence of the Ph chromosome
   - RNA analysis to demonstrate the presence of the BCR ABL gene product.
3. Other blood tests
   - Blood LDH levels are elevated
   - Uric acid level may be high due to increased cell breakdown.

Prolymphocytic leukaemia

1. This is a variant of chronic lymphatic leukaemia found mainly in males over the age of 60 years

2. 25 % of cases are of the T cell variety
3. There is massive splenomegaly with little lymphadenopathy
4. A very high leucocyte count, often in excess of 400 x 10^9/L
5. The characteristic cell is a large lymphocyte with a prominent nucleolus.
6. Treatment is generally unsuccessful and the prognosis very poor.
7. Leukapharesis, splenectomy and chemotherapy may be tried.

Hairy cell leukaemia

1. This is a rare chronic lymphoproliferative B cell disorder.
2. The male female ratio is 6:1
3. Median age at diagnosis is 50 years.
4. Presenting symptoms are those of general ill health and recurrent infection
5. Splenomegaly occurs in 90% but lymph node enlargement is unusual.
6. Severe neutropenia, monocytopenia and the characteristic hairy cells in the blood and bone marrow are typical.

Myelodysplastic syndrome (MDS)

1. This syndrome consists of a group of clonal haematopoietic disorders which represent steps in the progression to the development of leukaemia.
2. It presents with consequences of bone marrow failure (anaemia, recurrent infections or bleeding), usually in older people.
3. Median age at diagnosis is 69 years.
4. The blood film is characterised by cytopenias and abnormal-looking (dysplastic) blood cells, including macrocytic red cells and hypogranular neutrophils with nuclear hyper- or hyposegmentation.
5. The bone marrow is hypercellular with dysplastic changes in all three cell lines.
6. Blast cells may be increased but do not reach the 20% level which indicates acute leukaemia.
7. Chromosome analysis frequently reveals abnormalities, particularly of chromosome 5 or 7.

Thrombocytopenia

Clinical Significance

Determination of platelets is requested in the investigation of bleeding disorders.

Thrombocytopenia (decreased platelet count)- The count is less than 100,000 /cmm

The Mechanism leading to Thrombocytopenia

1. This may be due to decreased production of the bone marrow. This may be due :
   A. Bone marrow failure.
   B. Infiltration of the bone marrow by tumors or fibrosis.
2. Destruction or sequestration of the platelets by hypersplenism.
3. Antibodies destroying the platelets.
4. Destruction of the platelets by infection or drugs.
5. Increased utilization of disseminated intravascular coagulopathy.
6. In severe hemorrhage which leads to loss of platelets.
7. A large blood transfusion leads to a dilutional effect.

Causes of Thrombocytopenia

Physiological- New born, menstruating female

Pathological

- It is often associated with prolonged bleeding and poor clot retraction.
- It also occurs in aplastic anemia, pernicious megaloblastic hemolytic anemia
- Immune thrombocytopenia (ITP).
- Immune thrombocytopenic purpura (ITP)
- Hypersplenism.
- After a massive blood transfusion
- Infection like viral, and bacterial like HIV infection, dengue, dengue haemorrhaging fever, rubella infection
- Chemotherapy treatment.
- Leukemias, carcinoma, and myelofibrosis. This is due to the infiltration of the bone marrow.

- Disseminated intravascular coagulation (DIC).
- Toxemia of pregnancy, eclampsia.
- Antiplatelets antibody.
- Renal failure.
- Inherited diseases like Wiskott-Aldrich syndrome.
- An autoimmune disease like systemic lupus erythematosus

Thrombocytosis (increased platelet count)- The count is more than 400,000 /cmm

Physiological- At high altitude, during severe exercise

Pathological

Secondary Thrombocytosis

1. Platelets are acute-phase reactants; therefore, platelet counts increase in response to various stimuli, including systemic infections, inflammatory conditions, bleeding, and tumors. This phenomenon is called reactive or secondary thrombocytosis, which is a benign form of thrombocytosis
2. Malignant tumors like leukemia, chronic myelogenous leukemia and lymphoma,
3. Polycythemia vera.
4. Iron deficiency anemia.
5. Autoimmune diseases like Rheumatoid arthritis, and Systemic lupus erythematosus (SLE).
6. Hodgkin's lymphoma
7. Chronic pancreatitis and inflammatory bowel disease.
8. Tuberculosis.
9. Infection and Inflammatory disorders
10. Postsplenectomy or hyposplenism
11. Trauma
12. Chronic inflammatory conditions
13. Hemorrhage, blood loss, or both
14. Rebound thrombocytosis
15. Asplenia (anatomic or functional)
16. Electric shock
17. Idiopathic

Causes of essential thrombocytosis (primary thrombocythemia)

- The etiology and predisposing factors for the development of essential thrombocytosis (primary thrombocythemia) remain unclear.
- Genetic transmission of this disorder is rare, although reports show several families with multiple members affected by essential thrombocytosis (primary thrombocythemia).
- Research suggests that a thrombopoietin production or receptor abnormality can cause familial essential thrombocytosis (primary thrombocythemia).

Thrombocythemia - When the count is above one million /cmm.

Causes

- Essential thrombocythemia is a type of chronic myeloproliferative disorder.
- It's not clear what causes this condition. About 90% of people with the disorder have a gene mutation contributing to the disease.

If an underlying condition such as an infection or iron deficiency causes a high platelet count, it's called secondary thrombocytosis. Compared with essential thrombocythemia, secondary thrombocytosis causes less risk of blood clots and bleeding.

MCQs

1. Which of the following is NOT a cause of microcytic anemia?
   a. Thalassemia
   b. Anemia of chronic disease
   c. Iron deficiency anemia
   d. Pancytopenia
2. Select the following that enhance Fe absorption (select all that apply)
   a. Citric acid b. Polyphenols (tea)
   c. Phytate (bran) d. Calcium
3. How would you diagnose aplastic anemia?
   a. Blood smear
   b. Bone marrow biopsy
   c. Spleen biopsy
   d. CBC

4. Most cases of acute leukaemia in children are of which type:
   a. T-cell ALL
   b. Mature B-cell ALL
   c. Hairy cell leukaemia
   d. Pre-B-cell ALL

5. Point mutation is characterized by which of the following disorders?
   a. Thalassemia
   b. Sickle cell anaemia
   c. Night blindness
   d. Down's syndrome

6. Beta-thalassemia is caused by which chromosomal mutation?
   a. 10 b. 21
   c. 13 d. 11

Answer

1.	d	2.	a	3.	b
4.	d	5.	b	6.	d

CHAPTER

23

Hridroga

Definition of Hṛdroga

Diseases of Heart. *Hṛdroga* has been classified into five groups viz. *Vātika, Paittika, Kaphaja, Sannipātika,* and *Kṛmija.* One of the pathological conditions and syndromes identified by *Āyurveda.* (*C.S. Su.* 7. 22, 17. 52, 19. 3). *Ban. hṛdrog, Guj. dil nā rog, Hin. hridrog, Kan. Hṛdya rog, Mal. hridrogam, Mar. Hṛdyarog, Tam. īdya noya, Tel. gunde rog mūla.*

हेतु (Hetu)

व्यायामतीक्ष्णातिविरेकवस्तिचिन्ताभयत्रासगदातिचाराः ।
छर्द्यामसंधारणकर्शनानि हृद्रोगकर्तॄणि तथाऽभिघातः ॥
(*C.S.Ci. 26/77*)

वेगाघातोष्णारूक्षान्नैरतिमात्रोपसेवितैः ।
विरुद्धाध्यशनाजीर्णैरसात्म्यैश्चापि भोजनैः ॥
(*S.S.Utt. 43/3*)

Etiological factors of the disease *Hṛdroga* are as follows:- Excessive exercise, excessive use of sharp substances, excessive administration of purgation & enema, excessive worry, fear, terror, improper management of diseases, suppression of vomiting & *Āma,* emaciation & injury, suppression of natural urges, consumption of food which is hot & dry in excess quantity, use of incompatible foods, eating more quantity of food often, uncooked & unaccustomed food in more quantity.

हृद्रोग संप्राप्ति (Samprāpti of Hṛdroga)

दूषयित्वा रसं दोषा विगुणा हृदयं गताः ।
कुर्वन्ति हृदये बाधां हृद्रोगं तं प्रचक्षते ॥
(*S.S.Utt. 50/4*)

Due to above etiological factors *Doṣās* become aggravated and vitiate the *Rasa* present in the heart and gives rise to pain in the heart is known as *Hṛdroga.*

Gives rise to pain in the heart due to aggravation of Vāta

Hṛdroga Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kaphadi doṣās*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Localisation of *Kaphadi doṣās* in the *Hṛdaya* (heart) and vitiate the *Rasa dhātu*

↓

Gives rise to pain in the heart due to aggravation of *Vāta*

↓

Hṛdroga

हृद्रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Hṛdroga)

Doṣa	:	*Tridoṣaja* *Vāta* → *Vyāna, Prāṇa* *Pitta* → *Sādhaka* *Kapha* → *Kledaka, Avalambaka*
Dūṣya	:	*Dhātu* → *Rasa, Rakta* *Upadhātu* → *Tvacā* *Śārīrika Mala* → *Puriṣa* *Dhātu Mala* → *Kapha, Pitta*
Agni	:	*Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi	:	*Mandāgni*
Āma	:	*Āgnijanya*
Srotas	:	*Rasavaha, Prāṇavaha*
Srotoduṣṭi	:	*Saṅga & Vimārgamana*
Sancārasthāna	:	*Rasavaha Sroto Avayava, Raktavāhinyaḥ*
Vyaktasthāna	:	*Hṛdaya*
Udbhavasthāna	:	*Hṛdaya*
Adhiṣṭhāna	:	*Hṛdaya*
Svabhāva	:	*Āśukāri*
Prabhāva	:	General prognosis rule applicable
Rogamārga	:	*Madhyama*

सामान्य लक्षण (Sāmānya Lakṣaṇa)

वैवर्ण्यमूर्च्छाज्वरकासहिक्काश्वासास्यवैरस्यतृषाप्रमोहाः । छर्दिः
कफोत्क्लेशरुजोऽरुचिश्च हृद्रोगजाः स्युर्विविधा- स्तथाऽन्ये ॥
(C.S.Ci. 26/78)

General signs & symptoms of *Hṛdroga* are as follows:- Discolouration of the skin, fainting, fever, cough, hiccough, dyspnoea, bad taste in mouth, thirst, unconsciousness, vomiting, nausea, pain, anorexia & other various symptoms arise in heart disease.

हृद्रोग भेद (Hṛdroga Bheda)

चतुर्विधः स दोषैः स्यात् क्रिमिभिश्च पृथक्-पृथक् ।
(S.S.Utt. 3/5)

Hṛdroga is classified into four varieties i.e. *Vātaja, Pittaja, Kaphaja & Sannipātaja. Caraka* classified *Hṛdroga* into five varieties i.e. *Vātaja, Pittaja, Kaphaja, Sannipātaja & Krimija.*

वातज हृद्रोग (Vātaja Hṛdroga)

शोकोपवासव्यायामरूक्षशुष्काल्पभोजनैः ।
वायुराविश्य हृदयं जनयत्युत्तमां रुजम् ॥
वेपथुर्वेष्टनं स्तम्भः प्रमोहः शून्यता दरः ।
हृदि वातातुरे रूपं जीर्णे चात्यर्थवेदना ॥
(C.S.Su. 17/30-31)

आयम्यते मारुतजे हृदयं तुद्यते तथा ।
निर्मथ्यते दीर्य्यते च स्फोट्यते पाट्यतेऽपि च ॥
(S.S.Utt. 43/6)

हृच्छून्यभावद्रवशोषभेदस्तम्भाः समोहाः पवनाद्विशेषः।
(C.S.Ci. 26/79)

Vātaja Hṛdroga manifest due to grief, fasting, excessive exercise, intake of unctous, dry & inadequate quantity of food.

Due to above etiological factors *Vāta* gets aggravated & enters the heart and manifest *Vātaja Hṛdroga* & it is characterised by excruciating pain, trembling, cramps, stiffness, unconsciousness, vacant look, tearing pain, severe pain after the digestion of food, vacantness in heart, palpitations, wasting in cardiac area, tearing, obstruction in the heart, patient feels pain such as expanding, pricking, twisting, tearing, exploding or splitting.

पित्तज हृद्रोग (Pittaja Hṛdroga)

उष्णाम्ललवणक्षारकटुकाजीर्णभोजनैः ।
मद्यक्रोधातपैश्चाशु हृदि पित्तं प्रकुप्यति ॥
हृद्दाहस्तिक्तता वक्त्रे तिक्ताम्लोद्गिरणं क्लमः ।
तृष्णा मूर्च्छा भ्रमः स्वेदः पित्तहृद्रोगलक्षणम् ॥
(C.S.Su. 17/32-35)

पित्तात्तमोदूयनदाहमोहाः संत्रासंतापज्वरपीतभावाः ॥
(C.S.Ci. 26/79)

तृष्णोषादाहचोषाः स्युः पैत्तिके हृदयक्लमः ।
धूमायनञ्च मूर्च्छा च स्वेदः शोषो मुखस्य च ॥
(S.S.Utt. 43/7)

Pittaja Hṛdroga manifest due to consumption of hot, sour, salty, alkaline, acidic & heavy substances; excess intake of wine products; excess anger & exposure to sun.

Due to above etiological factors *Pitta* gets aggravated in the *Hṛdaya* & manifest symptoms like heart burn, bitter taste in the mouth, eructations with bitter & sour taste, exhaustion, thirst, unconsciousness, giddiness, perspiration, feeling of darkness, *Dūyana* (giddiness), burning sensation, unconsciousness, *Santrāsa* (distress), *Tāpa* (warmness), fever, yellowish discolouration, burning sensation all over the body, sucking pain, exhaustion of the heart, feeling of hot fumes coming out, fainting, perspiration & dryness of mouth.

कफज हृद्रोग (Kaphaja Hṛdroga)

अत्यादानं गुरुस्निग्धमचिन्तनमचेष्टनम् ।
निद्रासुखं चाभ्यधिकं कफहृद्रोगकारणम् ॥
हृदयं कफहृद्रोगे सुप्तं स्तिमितभारिकम् ।
तन्द्रारुचिपरीतस्य भवत्यश्मावृतं यथा ॥
(C.S.Su. 17/34-35)

स्तब्धं गुरु स्यात् स्तिमितं च मर्म कफात् प्रसेकज्वरकासतन्द्राः।
(C.S.Ci. 26/80)

गौरवं कफसंस्रावोऽरुचिः स्तम्भोऽग्निमार्दवम् ।
माधुर्य्यमपि चास्यस्य बलासावतते हृदि ॥
(S.S.Utt. 43/8)

Kaphaja Hṛdroga manifest due to consumption of excess food, intake of heavy & unctous food, little mental & physical exercise and excess sleep.

Due to above etiological factors *Kapha* gets aggravated & manifest *Kaphaja Hṛdroga* & it is characterised by numbness, stiffness & heavyness in heart; drowsiness, anorexia, weight in the cardiac region as if it is pressed with stone, excessive salivation, fever, cough, weakness of *Agni* & sweet taste in mouth.

सान्निपातिक हृद्रोग (Sānnipātika Hṛdroga)

हेतुलक्षणसंसर्गादुच्यते सान्निपातिकः ।
हृद्रोगः कष्टदः कष्टसाध्य उक्तो महर्षिभिः ।
(C.S.Su. 17/36)

विद्यात्त्रिदोज्ञं त्वपि सर्वलिङ्गं ॥
(C.S.Ci. 26/80)

Sānnipātika Hṛdroga manifest due to adoption of etiological factors which aggravates all the three *Doṣās* and manifest symptoms of all the three types of heart diseases. It is painful & difficult to cure.

कृमिज हृद्रोग (Kṛmija Hṛdroga)

त्रिदोषजे तु हृद्रोगे यो दुरात्मा निषेवते ॥
तिलक्षीरगुडादीनि ग्रन्थिस्तस्योपजायते ।
मर्मैकदेशे ते जाताः सर्पन्तो भक्षयन्ति च ॥
संङ्केदात् क्रिमयश्चास्य भवन्त्युपहतात्मनः ।
छिद्यमानं यथा शस्त्रैर्जातकण्डूं महारुजम् ॥
तुद्यमानं स हृदयं सूची भिरिव मन्यते ।
छिद्यमानं यथां शस्त्रै र्जात कण्डूं महारुजम् ॥
हृद्रोगं क्रिमिजं त्वेतैर्लिङ्गैर्बुद्ध्वा सुदारुणम् ।
त्वरेत जेतुं तं विद्वान् विकारं शीघ्रकारिणम् ॥
C.S.Su. 17/36-40

तीव्रातितोदं कृमिजं सकण्डूम्॥ *C.S.Ci. 26/80*

उत्क्लेशः ष्ठीवनं तोदः शूलो हल्लासकस्तमः ।
अरुचिः श्यावनेत्रत्वं शोषश्च कृमिजे भवेत् ॥
S.S.Utt. 43/9

Kṛmija Hṛdroga develops as an event of the heart disease caused by simultaneous vitiation of all the three *Doṣās* followed by consumption of sesamum, milk, jaggery etc. leading to development of nodules in the heart. *Rasadhātu* attains moistureness in heart & favours the development of *Kṛmi*, gradually spread to entire heart & eat away the heart.

Signs & Symptoms

Patient feels as if his heart is being pierced by needles or being cut into pieces by weapons, itching, pain, intense discomfort, acute condition, nausea, excess spitting, drowsiness, anorexia, blue colour of the eyes & emaciation. Considering the seriousness of this disease one should take immediate steps (emergency management).

उपद्रव (Upadrava)

भ्रमक्लमौ सादशोषौ ज्ञेयास्तेषामुपद्रवाः ।
कृमिजे कृमिजातीनां श्लैष्मिकाणाञ्च ये मताः ॥
(S.S.Utt. 43/10)

Complications of *Hṛdroga* are giddiness, exhaustion, debility & emaciation. *Kṛmija Hṛdroga* complications and *Kaphaja Kṛmi* complications are similar.

हृच्छूल (Hṛcchūla)

कफपित्तावरुद्धस्तु मारुतो रसमूर्च्छितः ।
हृदिस्थः कुरुते शूलमुच्छ्वासारोधकं परम् ॥
स हृच्छूल इति ख्यातो रसमारुतसम्भवः ॥
S.S.Utt. 42/131

Vāta getting obstruction in the chest by *Kapha* & *Pitta* and combine with *Rasa* in the heart & manifest symptoms like pain in chest, obstruction to breathing & it causes due to involvement of *Rasa* & *Vāta*.

उपशय/पथ्य (Upaśaya/Pathya)

The following measures and products are considered to be good and favourable in curing *Hṛdroga*. *Svedana Karma*, *Virecana*, *Vamana*, *Laṅghana* (fasting), *Basti Karma*, *Vilepī*, old red rice, meat soup of wild animals and birds, *Mudga*, *Yūṣa* of *Kulattha*, *Rāgā*, *Khaḍayūṣa*, *Kāmblika Yūṣa*, *Ṣāḍava*, fruit of *Abhaya*, *Paṭola*, banana fruit, old *Kuṣmāṇḍa*, sweet mango, sweet pomegranate, leaves of *Āragvadha*, new radish, *Eraṇḍa* oil, rain water, rock salt, grapes, buttermilk, old jaggery, dried ginger, caraway seeds, garlic, *Haraḍa*, *Kuṭaja*, coriander, pepper, ginger, *Sauvīra* (*Kānji*), *Śukta* (vinegar), honey, liquor, *Kastūrī*, white sandalwood, tamarind and *Tāmbūla*. (B.R. 33/78-81).

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures and items are strictly prohibited for persons suffering from *Hṛdroga*:- Obstructing the flow of *Tṛṣṇā*, *Vamana*, *Mūtra*, *Adhovāta*, *Śukra*, *Kāsa*, belching, heavy breathing caused due to hard work, stool and tears, water from *Sahyādri* and *Vindhyādri* rivers, sheep milk, contaminated water, *Kaṣāya Rasa*, incompatible food, foods containing hot, pungent and citric (*Āmla*) properties, edibles that are heavy to digest, old leaves, *Kṣāra* products, *Madhūka*, brushing teeth with wood sticks and blood letting. (B.R. 33/ 82-83).

MCQs

1. Number of types of *Hridroga* as per *Sushruta*
   a. 3 b. 4
   c. 5 d. 6
2. *Chikitsetamayamkhoram tam shikramshikrakarinam* (चिकित्सेदामयं घोरं तं शीघ्रं शीघ्रकारिणम्।) is about
   a. *Mahahikka* b. *Sanyasa*
   c. *Krimija hridroga* d. *Asmari*
3. *Krakachenaevadaryate* (क्रकचेनेव दार्यते) seen in which *Hridroga*
   a. *Krimija* b. *Tridoshaja*
   c. *Vatika* d. *Paittika*
4. Which among the following is not the *Upadrava* of *Hridroga*
   a. *Klama* b. *Sada*
   c. *Bhrama* d. *Jwara*
5. *Hridayaklama* is the feature of
   a. *Vatika Hridroga*
   b. *Paittika Hridroga*
   c. *Tridoshaja Hridroga*
   d. *Krimija Hridroga*
6. *Syavanetrata* is the feature of
   a. *Vatika Hridroga*
   b. *Paittika Hridroga*
   c. *Tridoshaja Hridroga*
   d. *Krimija Hridroga*
7. *Atyushna-kashaya-tikta-bhojana* is the *nidana* of
   a. *Pandu* b. *Rakta pitta*
   c. *Hridroga* d. *Mutrakrichra*
8. The *nidana* as in *Gulmanidana* results in which disease
   a. *Udara* b. *Arsas*
   c. *Hridroga* d. *Anaha*
9. *Sabdaasahishnuta* is the feature of
   a. *Rasa kshaya*
   b. *Vatajahridroga*
   c. *Krimijahridroga&Rasakshaya*
   d. *Rasa kshaya &Vataja hridroga*
10. Bharikam cha ashmagarbhavat is the feature of
   a. *Jalodara*
   b. *Baddodara*
   c. *Kaphaja hridroga*
   d. *Raktagulam*

Answer

1.	b	2.	c	3.	a
4.	d	5.	b	6.	d
7.	c	8.	c	9.	d
10.	c				

CHAPTER 24

Shotha/Shvayathu Nidana

Synonyms : शोफ (Śopha), शोथ (Śotha)

Introduction

Sotha is the diseases of *Udakavaha srotas* & it may occur as a disease or as a symptom in other diseases. *Acarya Caraka* explained *Śotha* chapter after *Kṣatakṣīṇa* because of their resemblance in causative factor i.e. *Marmopaghāta* (injury to vital parts). *Vāgbhaṭa* described *Śotha* along with *Pāṇḍuroga*, because *Śotha* is the principal complication of *Pāṇḍuroga*. *Śopha*, *Śothaka* and *Śvayathu* are the synonyms of *Śotha*.

Petting on pressure, oedema, heaviness in the body, horripilation, discolouration of the skin are the main signs and symptoms of *Śopha*. Its common varieties are as follows: *Vātaja Śopha* (*Hṛdjanya Śopha*). - This type of oedema is produced by the diminished pumping power of the heart. The oedema first appears on the feet, and increases during the day and tends to subside in the night. On pressing, the related portion of skin gets depressed, however, immediately after releasing the pressure, it regains its position. *Pittaja Śopha* (*Yakṛtjanya Śopha*) - The oedema due to the derangement of *Yakṛt* (liver) is identified as *Pittaja Śopha*. It first appears on the abdomen and subsequently it spreads up gradually. *Kaphaja Śopha* (*Vṛkkajanya Śopha*) - Oedema produced by the impaired function of kidneys has been identified as *Kaphaja Śopha*. It first appears in the pits of the eyes and subsequently spread to the other parts of the body.

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣā)

शोथः शवतीति । शु गतौ + बाहुलकात् । *Śabdakalpdruma*

शोफः शु गतौः बाहुलकात् । *Śabdakalpdruma*

श्वयथु श्वयतीति । श्वि गतिवृद्धोः । *Śabdakalpdruma*

The word *Śotha* and *Śopha* are derived from the root '*Śu*' which indicates "*Gatau*" which means to go and the word *Śvayathu* is derived from the root *Śvi* which denotes *Gativṛddhoḥ* which means to swell.

उत्सेधलिङ्गं श्वयथुं करोति । *Ci. 12/8 (Cakrapāṇi)*

Swelling or protuberance in any part of the body is called *Śvayathu.*

सर्वमुत्सेधं शोथमाहुरित्यर्थः ॥ *M.Ni. 36/1-2 (Madhu)*

Elevation or protuberance in any part of the body or whole body is known as *Sotha.*

उत्सेधं संहतं शोफं तमाहुर्निचयादतः ॥
A.H.Ni. 13/21

उत्सेधं संहतं शोथं तमाहुर्निचयादतः ॥
M.Ni. 36/2

That which is associated with Swelling or protuberance in any part of the body is called *Śotha* or *Śopha.*

रोग भेद (Roga bheda)

Classification of *Śvayathu* according to *Caraka* *(C.S.Ci. 12/4)*

Three types based on *Doṣic* predominance

वातज श्वयथु (*Vātaja Śvayathu*)
पित्तज श्वयथु (*Pittaja Śvayathu*)
कफज श्वयथु (*Kaphaja Śvayathu*)

It is also classified into

A. Exogenous
B. Endogenous

It is also classified into

A. एकाङ्गज (*Ekāṅgaja*)
B. सर्वज (*Sarvaja*)

आगन्तुहेतुः त्रिविधो निजश्च सर्वार्धगात्रावयवाश्रितत्वात् ॥
C.S.Ci. 12/7

Both exogenous and endogenous types of *Śvayathu* is classified into three types according to their location i.e. 1. *Śvayathu* pervading the whole body, 2. *Śvayathu* pervading the half of the body and 3. *Śvayathu* pervading to only one limb or organ.

सर्वसरस्तु पञ्चविधः, तद्यथा–वातपित्तश्लेष्मसन्निपातविषनिमित्तः ॥ *S.S.Ci. 23/3*

Sarvasara Śopha is classified into five varieties namely *Vātaja, Pittaja, Śleṣmaja, Sannipātaja* and *Viṣaja.*

निज श्वयथु हेतु एवं संप्राप्ति (Nija Śvayathu Hetu evam Samprāpti)

तत्रापतर्पितस्याध्वगमनादतिमात्रमभ्यवहरतो वा पिष्टान्नहरितकशाकलवणानि क्षीणस्य वाऽतिमात्रमम्लमुपसेवमानस्य मृत्पक्वलोष्टकटशर्करानूपौदकमांससेवनादजीर्णिनो वा ग्राम्यधर्मसेवनाद्विरुद्धाहारसेवनाद् वा हस्त्यश्वोष्ट्ररथपदातिसङ्क्षोभणादयोसितस्य दोषा धातून् प्रदूष्य श्वयथुमापादयन्त्यखिले शरीरे ॥
S.S.Ci. 23/4

One who is malnourished, who resort to long distance walk or consume foods in large quantities or consume food prepared from flour of grains, leafy vegetables and salty foods; or the person who is emaciated consuming food which is very sour, baked on mud plates or stones, foods with more of *Kaṭaśarkara* (lime stone vis-a-vis slaked lime), meat of animals of marshy lands, or one who is suffering from indigestion, or sexual intercourse (excessive or improper), intake of incompatible foods or excessive fatigue by riding on elephant, horse, camel, chariots, cavalry etc.

Due to above etrological factors *doṣās* gets aggravated and brings abnormality in *dhātus* leading to development of *Śvayathu* all over the body.

शुद्धामयाभक्तकृशाबलानां क्षाराम्लतीक्ष्णोष्णगुरूपसेवा ।
दध्याममृच्छाकविरोधिदुष्टगरोपसृष्टांन्ननिवेषणं च ॥
अर्शांस्यचेष्टा न च देहशुद्धिर्मर्मोपघातो विषमा प्रसूतिः ।
मिथ्योपचारः प्रतिकर्मणां च निजस्य हेतुः श्वयथोः प्रदिष्टः॥
C.S.Ci. 12/5-6

Consumption of *Kṣāra, Āmla, Tīkṣṇa, Uṣṇa* and *Guru* by a person who has become emaciated and debilitated due to evacuation, diseases and undernutrition; excessive use of curd, uncooked food, mud, pot herbs, antagonistic, defective and poisoned food, suffering from piles, lack of exercise, not undergoing biopurificatory therapies in appropriate time, injury to vital organs, difficult labour and faulty management of biopurificatory therapies.

आगन्तुज श्वयथु हेतु (Āgantuja Śvayathu Hetu)

बाह्यास्त्वचो दूषयिताऽभिघातः काष्ठाश्मशस्त्राग्निविषायसाद्यैः आगन्तु हेतुः । C.S.Ci. 12/7

Affliction of the external skin by the wooden piece, stone, sharp instruments, fire, poison and iron made articles etc. gives rise to *Āgantuja Śvayathu.*

श्वयथु निदान संप्राप्ति (Samprāpti of Śvayathu Nidāna)

बाह्याः सिराः प्राप्य यदा कफासृकपित्तानि संदूषयतीह वायुः । तैर्बद्धमार्गः स तदा विसर्पन्नुत्सेधलिङ्गं श्वयथुं करोति ॥
उरःस्थितैरूर्ध्वमधस्तु वायोः स्थानस्थितैर्मध्यगततैस्तु मध्ये ।
सर्वाङ्गः सर्वगतैः क्वचित्स्थैर्दोषैः क्वचित् स्याछ्वयथुस्तदाख्यः॥ *C.S.Ci. 12/8-9*

Due to above etiological factors *Vāta* reaches the external channels and affects *Kapha, Rakta* and *Pitta.* As a result of these factors, the passage gets obstructed which spreads to the nearby areas leading to development of oedema characterised by protuberance.

Doṣās seated in the chest cause swelling in upper part of the body, if these afflictions take place in the *pakvāśaya,* which is the location of *Vāta,* then swelling occurs in the lower parts of the body, if these afflictions take place in the middle of the body, then swelling occurs in the middle part of the body, when generalised they cause swelling all over the body and when located in any particular organ they cause localised swelling there itself and it is known by the respective organs.

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
Reaches the external channels
↓
Affects *Kapha, Rakta* and *Pitta.*
↓
obstruction in the *Srotas*
↓
which spreads to the nearby areas
↓
Leading to development of *Śvayathu*
(Oedema)
(Characterised by protuberance)

Pathogenesis of Śvayathu (Oedema)-Upper body parts

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
Reaches the channels of chest
↓
Affects *Kapha, Rakta* and *Pitta.*
↓
obstruction in the *Srotas*
↓
which spreads to the upper part of the body
↓
Leading to development of *Śvayathu* (Oedema) in the upper body parts

Pathogenesis of Śvayathu (Oedema)-Lower body parts

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
Reaches the channels of *Pakvāśaya,*
↓
Affects *Kapha, Rakta* and *Pitta.*
↓
obstruction in the *Srotas*
↓
which spreads to the lower part of the body
↓
Leading to development of *Śvayathu* (Oedema) in the lower body parts

Pathogenesis of Śvayathu (Oedema)-Middle body parts

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
Reaches the channels of middle of the body
↓
Affects *Kapha, Rakta* and *Pitta.*
↓
obstruction in the *Srotas*
↓
which spreads to the middle part of the body
↓
Leading to development of *Śvayathu* (Oedema) in the middle body parts

Pathogenesis of Śvayathu (Oedema)-Whole body parts/Generalised swelling

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
Reaches the channels of whole body
↓
Affects *Kapha, Rakta* and *Pitta.*
↓
Obstruction in the *Srotas*
↓
which spreads to all the body parts
↓
Leading to development of *Śvayathu* (Oedema) in the whole body parts (generalised swelling)

श्वयथु निदान संप्राप्ति घटक (Samprāpti Ghaṭaka of Śvayathu Nidāna)

Doṣa : *Tridoṣaja*

सर्वस्त्रिदोषोऽधिकदोषलिङ्गैस्तच्छब्दमभ्येति भिषग्जितं च ॥
C.S.Ci. 12/10

Vāta → Vyāna
Pitta → Pācaka
Kapha → Kledaka

Dūṣya : *Dhātu → Rasa, Rakta*
Upadhātu → Śirā, Tvacā
Śāririkamala →
Dhātumala →

Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*

Agniduṣṭi : *Mandāgni*

Āma	: *Agnijanya*
Srotas	: *Raktavaha, Rasavaha, Udakavaha*
Srotoduṣṭi	: *Saṅga*
Udbhavasthāna	: *Koṣṭha*
Vyaktasthāna	: *Tvacā, Māmsa*
Sancārasthāna	: *Bāhya Sirāḥ*
Svabhāva	: *Cirakāri* & *Āśukāri*
Prabhāva	: *Ekadoṣaja*, newly manifested and in strong person → curable otherwise incurable.
Rogamārga	: *Bāhya* & *Abhyantara*
Rogabheda	: 5 types, 2 types, 3 types.

पूर्वरूप (Pūrvarupa)

ऊष्मा तथा स्याद्दवथुः सिराणामायाम इत्येव च पूर्वरूपम् ।
C.S.Ci. 12/10

Premonitory signs & symptoms of *Śvayathu* are as follows

Increase body temperature, burning sensation in the skin, eyes etc. and appearance of venous network.

सामान्य लक्षण (Sāmānya Lakṣaṇa)

सगौरवं स्यादनवस्थितत्वं सोत्सेधमूष्माऽथ सिरातनुत्वम् ।
सलोमहर्षाऽङ्गविवर्णता च सामान्यलिङ्गं श्वयथोः प्रदिष्टम् ॥
C.S.Ci. 12/11

General signs & symptoms of *Śvayathu* are as follows : Heaviness, instability, swelling, increased body temperature, thinning of vessels, horripitation and discolouration of body parts.

विशेष लक्षण (Viśeṣa Lakṣaṇa)

वातज श्वयथु (Vātaja Śvayathu)

चलस्तनुत्वक्परुषोऽरुणोऽसितः प्रसुप्तिहर्षार्तियुतोऽनिमित्ततः । प्रशाम्यति प्रोन्नमति प्रपीडितो दिवाबली च श्वयथुः समीरणात् ॥ *C.S.Ci. 12/12*

तत्र वातश्वयथुररुणः कृष्णो वा मृदुरनवस्थितास्तोदादयश्चात्र वेदनाविशेषाः। *S.S.Ci. 23/5*

Vātaja Śvayathu is diagnosed based on its unstable nature; skin becomes thin, rough and reddish or blackish discolouration; soft to touch, associated with numbness, horripilation and pain like pricking etc; subsides without any apparent cause; when pressed, the swelling disappears but it reappears after the pressure is withdrawn and it is aggravated during day time.

पित्तज श्वयथु (Pittaja Śvayathu)

मृदुः सगन्धोऽसितपीतरागवान् भ्रमज्वरस्वेदतृषामदान्वितः ।
य उष्यतेऽस्पर्शसहोऽक्षिरागकृत् स पित्तशोथो भृशदाहपाकवान् ॥
C.S.Ci. 12/13

पित्तश्वयथुः पीतः सरक्तो वा मृदुः शीघ्रानुसार्यूषादयश्चात्र वेदनाविशेषाः। *S.S.Ci. 23/5*

Pittaja Śvayathu is diagnosed based on its softness, odourness, black, yellow or red, spreads quickly associated with giddiness, fever, sweating, thirst, narcosis; has burning sensation, tenderness, redness of eyes, inflammation and suppuration.

कफज श्वयथु (Kaphaja Śvayathu)

गुरुः स्थिरः पाण्डुररोचकान्वितः प्रसेकनिद्रावमिवह्निमान्द्यकृत।
स कृच्छ्रजन्मप्रशमो निपीडितो न चोन्नमेद्रात्रिबली कफात्मकः॥
C.S.Ci. 12/14

श्लेष्मश्वयथुः पाण्डुः शुक्लो वा स्निग्धः कठिनः शीतो मन्दानुसारी, कण्ड्वादयश्चात्र वेदनाविशेषाः। *S.S.Ci. 23/5*

Kaphaja Śvayathu is diagnosed based on is heavyness, stableness, paleness, ass, yellowish white or whitish colour, unctous, hard, cold to touch, spreads slowly, itching associated with anorexia, excessive salivation, excessive sleep, vomiting and poor digestion; this type of *Śvayathu* takes long time to appear and its cure also takes a long time; when pressed and if the pressure is released, the pit doesn't get filled up and it is aggravated during night.

सन्निपातज श्वयथु (Sannipātaja Śvayathu)

सन्निपातश्वयथुः सर्ववर्णवेदनः। *S.S.Ci. 23/5*

Sannipātaja Śvayathu is diagnosed based on the observation of signs & symptoms of all the three *Doṣās* together.

विषज श्वयथु (Viṣaja Śvayathu)

विषनिमित्तस्तु गरोपयोगाद् दुष्टतोयसेवनात् प्रकुथितोदकावगाहनात् । सविषसत्त्वदिग्धचूर्णेनावचूर्णनाद्वा सविषसमूत्रपुरीषशुक्रस्पृष्टानां वा तृणकाष्ठादीनां संस्पर्शनात्, स तु मृदुः क्षिप्रोत्थानोऽवलम्बी चलोऽचलो वा दाहपाकरागप्रायश्च भवति ।
S.S.Ci. 23/5

Etiology

If manifest due to ingestion of *Gara Viṣa* (artificial/homicidal poison), drinking polluted water, bathing or immershing in contaminated water, smearing the ash of poisonous animals, coming in contact with urine, stool, semen of poisonous animals and poisonous grass or plants.

Signs & symptoms

It is soft to touch, develops quickly, is drooping downward, either movable or immovable, has burning sensation and ulceration generally.

द्विदोषज एवं त्रिदोषज श्वयथु (Dvidoṣaja evam Tridoṣaja Śvayathu)

नदानाकृतिसंसर्गाच्छ्वयथुः स्याद्द्विदोषजः।
सर्वाकृतिः सन्निपाताच्छोथो व्यामिश्रलक्षणः॥

C.S.Su. 18/10

Presence of symptoms of two *Doṣās* and of all the three *Doṣas* together are to be noticed in *Dvidoṣaja* and *Sannipātaja Śothās* respectively.

अभिघातज शोथ (Abhighātaja Śotha)

अभिघातेन शस्त्रादिच्छेदभेदक्षतादिभिः।
हिमानिलोदध्यनिलैर्भल्लातकपिकच्छुजैः॥
रसैः शूकैश्च संस्पर्शाच्छ्वयथुः स्याद्विसर्पवान्।
भृशोष्मा लोहिताभासः प्रायशः पित्तलक्षणः॥

M.Ni. 36/11-12

Assault by weapons causing cutting, breaking or tearing of the parts, assault by frost, breezed, cyclone; contact with fumes or oil of *bhallātaka* (marking nut) and fine thorns of the pod of *kapikacchu* (mucuna pruriens), irritating juices of herbs, etc., bring about an oedema which spreads quickly, associated with severe burning sensation, redness and other symptoms of *Pittavṛddhi.*

अवयवस्थित शोथ लक्षण (Avayavasthita Śotha Lakṣaṇa)

दोषाःश्वयथुमूर्ध्वं हि कुर्वन्त्यामाशयस्थिताः॥
पक्वाशयस्था मध्ये तु वर्चः स्थानगतास्तत्वधः।
कृत्स्नदेहमनुप्राप्ताः कुर्युः सर्वसरं तथा॥

S.S. Ci. 12/6-7

Doṣas residing in the stomach produce oedema in the upper part of the body, residing in the large intestine they produce oedema in the trunk, residing in the rectum they produce oedema in the lower parts and if they are circulating all over the body they produce oedema every where.

साध्यासाध्यता (Sadhyāsādhyatā)

कृशस्य रोगैरबलस्य यो भवेदुपद्रवैर्वा वमिपूर्वकैर्युतः। स हन्ति मर्मानुगतोऽथ राजिमान् परिस्त्रवेद्धीनबलस्य सर्वगः॥ अहीनमांसस्य य एकदोषजो नवो बलस्थस्य सुखः स साधने।

C.S.Ci. 12/15-16

The patient of *Śvayathu* succumbs to death because of the following : Development of *Śvayathu* in emaciated persons and who has become weak because of afflictions by diseases, observation of complications like vomiting etc.; if *Śvayathu* manifest in vital parts, having streaks generalised with discharge in weak person.

Śvayathu is curable in those individuals whose muscle tissue is not reduced, is caused by single *Doṣa,* newly manifested and in strong persons.

श्वयथुर्मध्यदेशे यः स कष्टः सर्वगश्च यः।
अर्धाङ्गेऽरिष्टभूतश्च यश्चोर्ध्वं परिसर्पति॥
श्वासः पिपासा दौर्बल्यं ज्वरश्छर्दिररोचकः।
हिक्कातीसारकासाश्च शूनं सङ्क्षेपयन्ति हि॥

S.S.Ci. 23/7-9

Presence of swelling in half of the body associated with fatal signs, that which spreads upwards, that accompanied with symptoms such as dyspnoea, severe thirst, debility, fever, anorexia, hiccup, diarrhoea and cough.

उपशय/पथ्य (Upaśaya/Pathya)

The following measures and products are considered to be beneficial if taken in regulated quantity for treating a patient of *Śotha*: *Samśodhana* (biopurificatory therapeutics), *Laṅgana* (fasting), blood letting, *Svedana* therapy, *Pariṣecana*, old *Śāli* rice, *Kulattha*, *Mudga*, meat of *Godha*, *Śallaka*, peacock, partridge, cock, *Lāva*, wild animals, tortoise and fish, old *Ghṛtam*, buttermilk, liquor, honey, *Āsava*, beans of *Niṣpāva*, *Kāravellaka*, red *Śobhāñjana*, mango, *Karkoṭa*, *Mānamūlam*, *Suvarcalā*, carrot, *Paṭola*, *Vetrāgra*, radish, *Punarnavā*, *Citraka*, *Pāribhadra*, *Śṛīparṇa*, leaves of *Neem* and *Kokilākṣa*, oil of

Eraṇḍa, *Kaṭukī*, *Haraḍa*, *Kṣāra* products, *Bhallātaka*, *Guggulu*, *Lauha Bhasma*, *Kaṭu* and bitter juices, pro-digestive foods, urine of cow, goat and buffalo, *Kastūrī*, *Śilājātu* and all those *Pathya* (measures and eatables) which have been recommended in treatment of *Pāṇḍu* and cauterization. (B.R. 42/198-203)

Cook old paddy and *Śāli* rice with decoction of *Daśamūla* and obtain their *Yūṣa* and *Yūṣa* containing *Yava* and rice grains. Consuming these preparations and taking products containing *Kaṭu* juice with little quantity of *Ghṛtam* is also considered to be helpful for the patient of *Śotha*. (B.R. 42/204)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

शोफिनः सर्व एव परिहरेयुरम्ललवणदधिगुडवसापय-स्तैलघृतपिष्टमयगुरूणि ॥ S.S.Ci. 23/10

Patients of all varieties of *Śvayathu* should avoid ingestion of foods which are sour or salty, curd, jaggery, fat, milk, oil and ghee, that containing more of corn flour and which are hard for digestion.

The following measures and products should be avoided by a patient of *Śotha*: continuous intake of contaminated air and water, obstructing the flow of body excretions, drinking incompatible liquids, incompatible foods, eating soil, meat of animals that live in villages or in marshy areas, salt, dried leaves, fresh rice, products of raw sugar (*Guḍa*), paste-based dishes, curd, *Kṛśara*, liquors without water, citrus products, fried paddy, dry meat, taking edibles that are heavy to digest, unfavorable and steaming food items, sleeping during day time and indulging in intercourse. (B.R. 42/205-206)

Products made up of pastes (of *Māṣa* and wheat), warm foods, salty, liquor, soil, day sleeping, meat of marshy animals and birds, water, jaggery, oil and sweet products are prohibited for the *Śotha* patient. (B.R. 42/207).

Differentiate between Nija Śvayathu & Āgantuja Śvayathu

S.No.	*Nija Śvayathu*	*Āgantuja Śvayathu*
Etiology	Consumption of *Kṣāra*, *Āmla*, *Tīkṣṇa*, *Uṣṇa* and *Gurū* by a person who has become emaciated and debilitated due to evacuation, diseases and under nutrition; excessive use of curd, uncooked food, mud, pot herbs, antagonistic, defective and poisoned food, suffering from piles, lack of exercise, not undergoing bio-purificatory therapies in appropriate time, injury to vital organs, difficult labour and faulty management of bio-purificatory therapies	Affliction of the external skin by the wooden piece, stone, sharp instruments, fire, poison and iron made articles etc. gives rise to *Āgantuja Śvayathu*.

Differentiate Doṣaja Śvayathu/Śotha

S.No.	*Vātaja Śvayathu*	*Pittaja Śvayathu*	*Kaphaja Śvayathu*	*Sannipātaja Śvayathu*
Clinical presentation	Skin becomes thin, rough and reddish or blackish discolouration; soft to touch, associated with numbness, horripilation and pain like pricking etc; subsides without any apparent cause; when pressed, the	Softness, odourness, black, yellow or red, spreads quickly associated with giddiness, fever, sweating, thirst, narcosis; has burning sensation, tenderness, redness of eyes, inflam-	Heaviness, stableness, paleness, ass, yellowish white or whitish colour, unctous, hard, cold to touch, spreads slowly, itching associated with anorexia, excessive salivation, excessive sleep, vomiting and poor digestion; this type	*Sannipātaja Śvayathu* is diagnosed based on the observation of signs & symptoms of all the three *Doṣās* together

	swelling disappears but it reappears after the pressure is withdrawn	mation and suppuration.	of *Śvayathu* takes long time to appear and its cure also takes a long time; when pressed and if the pressure is released, the pit doesn't get filled up	
Aggravation	It is aggravated during day time.		It is aggravated during night	

MCQs

1. *Kshiprothanasama* is the feature of
   a. *Vataja Shotha* b. *Paithika Shotha*
   c. *Kaphaja Shotha* d. *Abhighataja Shotha*
2. *Avalambhiseekhrodaharujakara*is
   a. *Abhighataja Shotha* b. *Agni visarpa*
   c. *Vishaja Shotha* d. *Kardamavisarpa*
3. The type of *Shotha* aggravates in night
   a. *Vataja Shotha* b. *Paithika Shotha*
   c. *Kaphaja Shotha* d. *Vishaja Shotha*
4. *Rathravalpe diva mahan* is the feature of which *sopha*
   a. *Vataja Shotha* b. *Paithika Shotha*
   c. *Kaphaja Shotha* d. *Vishaja Shotha*
5. *Bhallathaka, kapikachu* are considered as the *nidana* of which type of *Sopha*
   a. *Paithika Shotha* b. *Kaphaja Shotha*
   c. *Vishaja Shotha* d. *Abhighataja*
6. *Sthana* of *Sopha*
   a. *Twak Rakta*
   b. *Twak Rakta Mamsa*
   c. *Twak Mamsa*
   d. *Twak Mamsa Medas*
7. What is the number of types of Sopha as per Susruta
   a. 9 b. 6 c. 3 d. 2
8. दवथुःसिरायामोऽङ्गगौरवम् is the *Purvarupa* of
   a. *Sopha* b. *Rakta pitta*
   c. *Daha* d. *Pandu*
9. मृदुश्चलोऽवलम्बीचशीघ्रोदाहरुजाकरः is the feature of
   a. *Paithika Shotha* b. *Kaphaja Shotha*
   c. *Vishaja Shotha* d. *Abhighataja*
10. भृशोष्मालोहिताभासः प्रायशः पित्तलक्षणः is the feature of
   a. *Vishaja Shotha* b. *Agni visarpa*
   c. *Kardamavisarpa* d. *Abhighataja Shotha*

Answer

1.	a	2.	c	3.	c
4.	a	5.	d	6.	c
7.	b	8.	a	9.	c
10.	d				

CHAPTER

25

Cardiovascular Disorders

Coronary Artery Disease (Ischemic Heart Disease and Myocardial Infraction)

- It is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium
- It typically occurs when there is an imbalance between myocardial oxygen supply and demand.
- The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery.

Epidemiology and risk factors

- IHD causes more deaths and disability and incurs greater economic costs than any other illness in the developed world.
- Genetic factors, a high-fat and energy-rich diet, smoking, and a sedentary lifestyle are associated with the emergence of IHD
- Obesity, insulin resistance, and type 2 diabetes mellitus are increasing and are powerful risk factors for IHD.
- With urbanization in countries with emerging economies and a growing middle class, elements of the energy-rich Western diet are being adopted.
- In light of the projection of large increases in IHD throughout the world, IHD is likely to become the most common cause of death worldwide by 2020.

Pathophysiology

- Central to an understanding of the pathophysiology of myocardial ischemia is the concept of myocardial supply and demand
- By reducing the lumen of the coronary arteries, atherosclerosis limits appropriate increases in perfusion when the demand for flow is augmented, as occurs during exertion or excitement.
- When the luminal reduction is severe, myocardial perfusion in the basal state is reduced.
- Coronary blood flow also can be limited by spasm (see Prinzmetal's angina) arterial thrombi, and, rarely, coronary emboli as well as by ostial narrowing due to aortitis.
- Congenital abnormalities such as the origin of the left anterior descending coronary artery from the pulmonary artery may cause myocardial ischemia and infarction in infancy, but this cause is very rare in adults.
- Myocardial ischemia also can occur if myocardial oxygen demands are markedly increased and particularly when coronary blood flow may be limited, as occurs in severe left ventricular hypertrophy due to aortic stenosis. The latter can present with angina that is indistinguishable from that caused by coronary atherosclerosis largely owing to subendocardial ischemia
- A reduction in the oxygen-carrying capacity of the blood, as in extremely severe anemia or in the presence of carboxyhemoglobin, rarely causes myocardial ischemia by itself but may lower the threshold for ischemia in patients with moderate coronary obstruction.

Microvascular angina

- Abnormal constriction or failure of normal dilation of the coronary resistance vessels also can cause ischemia. When it causes angina, this condition is referred to as *microvascular angina.*

Coronary Atherosclerosis

- Epicardial coronary arteries are the major site of atherosclerotic disease.

Major risk factors

- High levels of plasma low-density lipoprotein (LDL)
- Low plasma high-density lipoprotein (HDL)
- Cigarette smoking
- Hypertension
- Diabetes mellitus

Pathogenesis

Risk factors

↓

Disturb the normal functions of the vascular endothelium

↓

These functions include local control of vascular tone, maintenance of an antithrombotic surface, and control of inflammatory cell adhesion and diapedesis.

↓

The loss of these defenses leads to inappropriate constriction, luminal thrombus formation, and abnormal interactions between blood cells, especially monocytes and platelets, and the activated vascular endothelium

↓

Functional changes in the vascular milieu ultimately result in the subintimal collections of fat, smooth muscle cells, fibroblasts, and intercellular matrix that define the atherosclerotic plaque

↓

This process develops at irregular rates in different segments of the epicardial coronary tree and leads eventually to segmental reductions in cross-sectional

area, i.e., plaque formation.

↓

Atherosclerosis

Stable Angina Pectoris

- Transient myocardial ischemia causes episodic clinical syndrome
- Males constitute 70% of all patients with angina pectoris and an even greater proportion of those less than 50 years of age.
- It is, however, important to note that angina pectoris in women is often atypical in presentation

History-clinical features

- The typical patient with angina is a man >50 years or a woman >60 years of age who complains of episodes of chest discomfort, usually described as heaviness, pressure, squeezing, smothering, or choking and only rarely as frank pain.

Levine's sign

- When the patient is asked to localize the sensation, he or she typically places a hand over the sternum, sometimes with a clenched fist, to indicate a squeezing, central, substernal discomfort
- Angina is usually crescendo-decrescendo in nature, typically lasts 2 to 5 min, and can radiate to either shoulder and to both arms (especially the ulnar surfaces of the forearm and hand).
- It also can arise in or radiate to the back, interscapular region, root of the neck, jaw, teeth, and epigastrium.
- Angina is rarely localized below the umbilicus or above the mandible.
- A useful finding in assessing a patient with chest discomfort is the fact that myocardial ischemic discomfort does not radiate to the trapezius muscles; that radiation pattern is more typical of pericarditis.
- Although episodes of angina typically are caused by exertion (e.g., exercise, hurrying, or sexual activity) or emotion (e.g., stress, anger, fright, or frustration) and are relieved by rest
- Pain may occur at rest- "Unstable Angina Pectoris,"
- While the patient is recumbent causes pain- Angina Decubitus.

Nocturnal angina

- The patient may be awakened at night by typical chest discomfort and dyspnea.
- Nocturnal angina may be due to episodic

tachycardia, diminished oxygenation as the respiratory pattern changes during sleep, or expansion of the intrathoracic blood volume that occurs with recumbency; the latter causes an increase in cardiac size (end-diastolic volume), wall tension, and myocardial oxygen demand that can lead to ischemia and transient left ventricular failure.

- The threshold for the development of angina pectoris may vary by time of day and emotional state.

Physical Examination

- Abdominal aortic aneurysm
- Carotid arterial bruits
- Diminished arterial pulses in the lower extremities.
- Xanthelasmas
- Xanthomas
- Pulse contour at multiple locations
- Comparing the blood pressure between the arms and between the arms and the legs (ankle-brachial index).
- Examination of the fundi may reveal an increased light reflex and arteriovenous nicking as evidence of hypertension.
- There also may be signs of anemia, thyroid disease, and nicotine stains on the fingertips from cigarette smoking.
- A protuberant abdomen may indicate that the patient has the metabolic syndrome and is at increased risk for atherosclerosis.

Palpation

- It may reveal cardiac enlargement and abnormal contraction of the cardiac impulse (left ventricular dyskinesia).
- Tenderness of the chest wall, localization of the discomfort with a single fingertip on the chest, or reproduction of the pain with palpation of the chest makes it unlikely that the pain is caused by myocardial ischemia.

Auscultation

- These auscultatory signs are best appreciated with the patient in the left lateral decubitus position.
- Auscultation can uncover arterial bruits, a third and/or fourth heart sound
- Apical systolic murmur due to mitral regurgitation.

Laboratory Examination

- Microalbuminuria -The urine should be examined for evidence of diabetes mellitus and renal disease

Examination of the blood

- Measurements of lipids (cholesterol—Total, LDL, HDL—and triglycerides)
- Glucose (hemoglobin A_{1C})
- Creatinine
- Hematocrit
- Thyroid function.

A chest x-ray

- A chest x-ray is important as it may show the consequences of IHD, i.e., cardiac enlargement, ventricular aneurysm, or signs of heart failure.

C-reactive protein (CRP)

- Evidence exists that an elevated level of high-sensitivity C-reactive protein (CRP) (specifically, between 0 and 3 mg/dL) is an independent risk factor for IHD

  The major benefit of high-sensitivity CRP is in reclassifying the risk of IHD in patients in the "intermediate" risk category on the basis of traditional risk factors

Congestive Cardiac failure

Congestive heart failure (CHF) is a common clinical syndrome, and conventionally implies right ventricular failure associated with left heart failure. However, the latter may be minimal or sometimes even absent as in chronic cor-pulmonale. It is usually a chronic condition but may occasionally be acute as with massive myocardial infarction, or following an abrupt increase in pre-existing heart failure. It may then become a medical emergency requiring prompt treatment.

Etiopathology

Nearly all cases of CHF have an obvious underlying disease involving the myocardium or the cardiac valves, the most frequent being rheumatic, ischemic or hypertensive heart disease, thyrotoxicosis, various forms of carditis and cardiomyopathy, and chronic cor-pulmonale. It is also associated with concomitant infection, acute massive hemorrhage, after surgical operation or from discontinuation of digitalis and diuretics. It is important to identify such precipitating causes both for immediate management as well as to prevent acute recurrence.

The pathophysiology of CHF is complex. It is certainly multifactorial, the two most significant factors being: (i) a decrease in cardiac output due to either impaired cardiac contraction or insufficient cardiac filling; and (ii) consequent adaptive measures of which activation of neuro-hormonal systems is specially important. This includes release of norepinephrine and activation of renin-angiotensin-aldosterone system. These adaptive mechanisms increase heart rate, blood and cardiac filling pressure, and cause redistribution of blood flow an attempt to maintain adequate overall pumping functions of the heart, and preserve perfusion of the vital organs. Frank heart failure occurs when the capacity of these adaptive mechanisms is superseded, and, depending upon the rapidity and severity the cardiac performance becomes impaired.

Clinical features

1. Fatigability and varying degree of breathlessness
2. Orthopnoea
3. Paroxysmal nocturnal dyspnea
4. Mild cyanosis
5. Tachycardia
6. Irregular pulse
7. Pulmonary rales- from the base upwards with the worsening of LHF.

O/E : Advanced cases invariably develop the classical triad of right heart failure viz., elevated jugular venous pressure, tender hepatomegaly and subcutaneous edema over dependent parts of the body. More severe cases may develop serous effusions and extensive edema involving the whole body. The blood pressure is usually low with a small pulse pressure.

MCQs

1. Abdominal aortic aneurysm, carotid arterial bruits, and diminished arterial pulses in the lower extremities indicates
   a. Atherosclerotic disease
   b. Aortic aneurysm
   c. Diabetes mellitus
   d. Peripheral arterial disease
2. Homan's sign is neither specific nor sensitive for diagnosis of
   a. Rheumatoid arthritis
   b. Muscular atrophy
   c. Primary neuromuscular disorder.
   d. Deep venous thrombosis
3. ———usually develops gradually with exertion, emotional excitement, or afterheavy meals.
   a. Stable angina pectoris
   b. Unstable Angina.
   c. Myocardial Infarction
   d. Pericarditis
4. Which of the following statements is CORRECT?
   a. Myocardial tissue necrosis occurs immediately when blood flow is occluded.
   b. Cardiac troponin is highest 30 minutes after infarction.
   c. Inflammatory biomarkers are often elevated after an acute MI.
   d. Chest pain is always present in myocardial infarctions.

Answer

1. a 2. d 3. a 4. c

CHAPTER 26

Kamala

Introduction

If a patient of *pāṇḍu roga* idulge in eatables and habits which promote *pitta,* his blood and muscle tissues get vitiated and this situation generate the disease (known as *kāmalā*).

Deep yellow colouration of the eyes, skin, nails and feces; yellow feces and urine, skin colour resembling that of frog (greenish, brown, or brownish yellow), weakness of sense organs, burning sensation, indigestion, weakness, debility, anorexia are the features of *kāmalā* (jundice). It involves both the *koṣṭha* (alimentary tract) and also the *śākhā* (*rakta* etc.) and other *dhātus* (tissues). *(CS. Ci.* 16. 34-36*)*.

If a patient of *pāṇḍu* resorts to *pitta*-aggravating regimens in excess, his blood and muscle tissue get afflicted by *pitta* to produce diseases. His eyes becomes excessively yellow and there wil be yellowness of the skin, nails and the face. The stool and the urine of the patient becomes either red or yellow in colour or like the colour of a frog (pale white). His sense organs get afflicted. He gets burning sensation, indigestion, weakness, prostration, anorexia and emaciation. This condition caused by excessive aggravation of *pitta* is called *kāmala* (jaundice). It is located either in *koṣṭha* (alimentary tract) or *śākhā* (tissues in the periphery) or both in *koṣṭha* and *śākhā*. *Bad-prognostic signs of Jaundice*. The patient of jaundice having red eyes and face, blood in vomiting, stool and urine, appearance of darkness before eyes, burning sensation, anorexia, thirst, *ānāha* (wind formation associated with constipation), drowsiness, delusion and loss of the power of digestion and unconsciousness succumbs to death immediately. Ban. *kāmlā*, Guj. *kāmḍo,* Hin. *pīliā*, Kan. *kāmāle, arisinmandige,* Mal. *manjppittam,* Mar. *kāvil, kāmiṇ,* Ori. *haldiā kāmlā* Pan. *pīliā*, Tam. *kanmalai, hanjlnoya*, Telg. *pasirikalu, kāmerlū*.

सकामलापानकिपाण्डुरोगः कुम्भाह्वयो लाघरकोऽलसाख्यः ।
विभाष्यते लक्षणमस्य कृत्स्नं निबोध वक्ष्याम्यनुपूर्वशस्तत् ॥

S.S.Utt. 44/6

Disease *Pāṇḍu roga* is called by various names like *Kāmalā, Pānaki, Pāṇḍuroga, Kumbha-kāmalā, Lāgharaka* and *Alasa.*

कामला निदान (Nidāna of Kāmalā)

यो ह्यामयान्ते सहसाऽन्नमम्लमद्यादपथ्यानि च तस्य पित्तम् ।
करोति पाण्डुं वदनं विशेषात् पूर्वैरितौ तन्द्रिबलक्षयौ च ॥

S.S.Utt. 44/10

पाण्डुरोगी तु योऽत्यर्थं पित्तलानि निज़ेवते ।

C.S.Ci. 16/34

भवेत्पित्तोल्बणस्यासौ पाण्डुरोगादृतेऽपि च ॥

A.H.Ni. 13/17

Consumption of *Pitta* aggravating etiological factors like sour foods, alcohol and unhealthy foods & activities just after recovery from *Pāṇḍu roga.*

If *Pāṇḍurogi* consumes excess *Pitta* aggravating factors. One who consumes *Pitta* aggravating factors in excess.

कामला संप्राप्ति (Samprāpti of Kāmalā)

तस्य पित्तमसृङ्मांसं दग्ध्वा रोगाय कल्पते ॥

C.S.Ci. 16/34

Aggravated *Pitta* burns the *Rakta* & *Māmsa* and manifest *Kāmala.*

Pathogenesis of *Paratantra Kāmalā*

Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Pittādi doṣās*

↓

Diminution of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Aggravated *Pitta* expelled from *Hṛdaya Via* ten blood vessels, by powerful *Vāta* circulates all over the body (afflicts *dhātus* mainly *Raktadhātu*) and it is also responsible for the less production of *Poṣaka* (nutrient portion) from the *Rasadhātu* as a result depletion of *Rakta* takes place

↓

It reaches the space between *Tvak* and *Māmsa*

↓

Brings laxity and heaviness in the *Kapha, Vāta, Rakta, Tvak* and *Māmsa*

↓

leading to abnormal complexion like pale yellow, deep yellow and greenish colouration.

↓

deficiency of *Rakta, Meda, Niḥsāra* (loss of essence) associated with improper functioning of sense organs and discolouration.

↓

Pāṇḍu roga

↓

Paratantra Kāmalā

Pathogenesis of Swatantra *Kāmalā*

Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Pittādi doṣās*

↓

Diminution of *Agni*

↓

Development of *Āma* inside the gastro intestinal tract

↓

Aggravated *Pitta* expelled from *Hṛdaya Via* ten blood vessels, by powerful *Vāta* circulates all over the body (afflicts *dhātus* mainly *Raktadhātu*) and it is also responsible for the less production of *Poṣaka* (nutrient portion) from the *Rasadhātu* as a result depletion of *Rakta* takes place

↓

It reaches the space between *Tvak* and *Māmsa*

↓

Aggravated *Pitta* burns the *Rakta* & *Māmsa*

↓

leading to abnormal complexion like pale yellow, deep yellow and greenish colouration.

↓

Svatantra Kāmalā

कामला संप्राप्ति घटक (Samprāpti Ghaṭaka of Kāmalā)

Doṣa : *Pitta Pradhāna Tridoṣaja*
Vāta → Samāna, Vyāna
Pitta → Pācaka, Ālacaka, Bhrājaka
Kapha → Kledaka
Dūṣya : *Dhātu→ Rasa, Rakta, Māmsa, Meda;* later all the *Dhātus (Niḥsāra)*
Updhātu →
Śārīrika Mala→ Mūtra, Puriṣa
Dhātu Mala→ Kapha, Pitta
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Rasavaha, Raktavaha, Annavaha*
Sroto duṣṭi : *Saṅga, Vimārgagamana*
Udbhavasthāna : *Āmāśayottha, Koṣṭha, Mahāsrotas*
Sancārasthāna : *Koṣṭha*
Vyaktasthāna : *Tvak, Nakha, Netra, Ānana, Koṣṭa, Śākhā*
Svabhāva : *Cirakāri*
Prabhāva : *Mentioned under prognosis*
Roga bheda : 2 types
1. कोष्ठाश्रय *(Koṣṭhāśraya)*
2. शाखाश्रय *(Śākhāśraya)*

Classification of *Kāmalā*

कामला बहुपित्तैषा कोष्ठशाखाश्रया मता ॥
C.S.Ci. 16/36

It is classified into two types : *Śākhāśraya Kāmala* may manifest without *Pāṇḍu roga,* if person consumes *Pitta* aggravating factors but in this type of *Kāmala, pitta* is less aggravated. That's why sour and pungent things are prescribed for the treatment of *Śākhāśraya Kāmalā* patients to bring the *Doṣās* from *Śākhā* to the *Koṣṭha*. However, *Koṣṭhāśraya Kāmalā* manifest due to excess aggravation of *Pitta (Bahu pitta)*.

According to *Hārita, Pāṇḍu roga* is classified into eight varieties namely *Vātaja, Pittaja, Kaphaja, Sannipātaja, Mṛd-bhakṣaṇaja, Śākhāśrita Kāmala, Koṣṭhāśrita Kāmalā* and *Halimaka*.

शाखाश्रय कामला/वातकफ प्रधान (Śākhāsraya Kāmalā/Vāta Kapha Pradhāna)

तिलपिष्टनिभं यस्तु वर्चः सृजति कामली ॥
श्लेष्मणा रुद्धमार्गं तत् पित्तं कफहरैर्जयेत् ।
रुक्षशीतगुरुस्वादुव्यायामैर्वेगनिग्रहैः ॥
कफसंमूर्च्छितो वायुः स्थानात् पित्तं क्षिपेद्बली ।
हारिद्रनेत्रमूत्रत्वक् श्वेतवर्चास्तदा नरः ॥
भवेत् साटोपविष्टम्भो गुरुणा हृदयेन च ।
दौर्बल्याल्पाग्निपार्श्वार्तिहिक्काश्वासारुचिज्वरैः ॥
क्रमेणाल्पेऽनुसज्येत पित्ते शाखासमाश्रिते ।

C.S.Ci. 16/124-128

Due to excessive consumption of rough, cold, heavy, sweet products, excessive exercise and suppression of natural urges leads to aggravation of both *Vāta* and *Kapha*. *Vāta amalgamates* with *Kapha* and expells the *Pitta* from its own site and manifest symptoms like yellowish discolouration of eyes, urine, and skin associated with whitish stools, gurgling sound in abdomen, constipation and heaviness in cardiac region. Patient passes stools having the colour of *Sesamum-paste* due to obstruction for the flow of *Pitta* to the *Koṣṭha* by *Kapha*.

Majority of the *Pitta* gets located in the *Śākhā* and there is reduction in the proportion *Pitta* in the *Koṣṭha* leads to weakness, *Agnimāndya,* pain on both sides of the chest, hiccup, dyspnoea, anorexia and fever.

That's why *Kapha* alleviating therapies are prescribed to pacify *Kapha* and simultaneously aggravating *Pitta*. Aggravation of *Pitta* is essential to bring back the *Pitta* from the *Śākhā* to the *Koṣṭha*.

कोष्ठाश्रय कामला/पित्त प्रधान (Koṣṭhāśraya Kāmalā/Pitta Pradhāna)

हारिद्रनेत्रः स भृशं हारिद्रत्वङ्नखाननः ।
रक्तपीतशकृन्मूत्रो भेकवर्णो हतेन्द्रियः ॥
दाहाविपाकदौर्बल्यसदनारुचिकर्षितः ।
कामला बहुपित्तैषा कोष्ठशाखाश्रया मता ॥

C.S.Ci. 16/35-36

Koṣṭhāśraya Kāmalā manifest due to excess aggravation of *Pitta* and it is characterised by deep yellowish discolouration of eyes, skin, nails and oral cavity; reddish-yellowish discolouration of stool and urine; appearance of whole body resembles like frog; impaired sensory functions associated with burning sensation, indigestion, debility, malaise and anorexia.

कुम्भ कामला (Kumbha Kāmalā)

कालान्तरात् खरीभूता कृच्छ्रा स्यात् कुम्भकामला ।
कृष्णपीतशकृन्मूत्रो भृशं शूनश्च मानवः ॥
सरक्ताक्षिमुखच्छर्दिविण्मूत्रो यश्च ताम्यति ।
दाहारुचितृषानाहतन्द्रामोहसमन्वितः ॥
नष्टाग्निसंज्ञः क्षिप्रं हि कामलावान् विपद्यते ।

C.S.Ci. 16/37-39

भेदस्तु तस्याः खलु कुम्भसाह्वः शोफो महांस्तत्र च पर्वभेदः ॥

S.S.Utt. 44/12

Improper treatment or delay in the management of *Kāmalā* leads to chronicity of the disease and it becomes difficult to cure because of its deep seated nature, are the characteristic feature of *Kumba Kāmalā*.

Signs and Symptoms

Blackish and yellowish discolouration of urine and stool; oedema all over the body; reddishness in eyes, oral cavity, vomiting materials, stools and urine; fainting; burning sensation, anorexia, thirst, hardness in bowels, drowsiness, confusion, sudden loss of power of agni and consciousness and pain in joints.

It is the advanced stage of *Koṣṭhāśraya Kāmalā*.

हलीमक कामला/वातपित्त प्रधान (Halīmaka Kāmalā/ Vāta-Pitta Pradhāna)

तं वातपित्ताद्धरिपीतनीलं हलीमकं नाम वदन्ति तज्ज्ञाः ॥
S.S.Utt. 44/14

यदा तु पाण्डोर्वर्णः स्याद्धरितश्यावपीतकः ॥
बलोत्साहक्षयस्तन्द्रा मन्दाग्नित्वं मृदुज्वरः ।
स्त्रीष्वहर्षोऽङ्गमर्दश्च श्वासस्तृष्णाऽरुचिर्भ्रमः ॥
हलीमकं तदा तस्य विद्यादनिलपित्ततः ।
C.S.Ci. 16/132-134

हरितश्यावपीतत्वं पाण्डुरोगे यदा भवेत् ॥
वातपित्ताद् भ्रमस्तृष्णा स्त्रीष्वहर्षो मृदुर्ज्वरः ।
तन्द्राबलानलभ्रंशो लोढरं तं हलीमकम् ॥
अलसं चेति शंसन्ति- *A.H.Ni. 13/19-20*

Halīmaka is the advanced stage of *Kāmalā* and manifest due to aggravation of *Vāta* and *Pitta.* It is characterised by greenish, blackish or yellowish discolouration associated with loss of strength and energy, drowsiness, *mandāgni,* mild fever, lack of interest towards women, malaise, dyspnoea, thirst, anorexia and giddiness. It is also known as *Loḍhara* and *Alasa.*

लाघरक एवं अलसाख्य (Lāgharaka evam Alasākya)

ज्वराङ्गमर्दभ्रमसादतन्द्राक्षयान्वितो लाघरकोऽलसाख्यः ॥
S.S.Utt. 44/12

These are the advanced stage of *Kāmalā* associated with fever, body aches, giddiness, debility, stupor and *Dhātukṣaya.*

पानकि (*Pānakī*)

सन्तापो भिन्नवर्चस्त्वं बहिरन्तश्च पीतता ।
पाण्डुता नेत्रयोर्यस्य पानकीलक्षणं भवेत् ॥
M.Ni. 8/24

It is the advanced stage of *Kāmalā* associated with fever, diarrhoea, yellowish discolouration internally and externally and whitish discolouration in the eyes.

उपद्रव (Upadrava)

उपद्रवास्तेष्वरुचिः पिपासा छर्दिर्ज्वरो मूर्द्धरुजाऽग्निसादः ।
शोफस्तथा कण्ङ्गतोऽबलत्वं मूर्च्छा क्लमो हृद्यवपीडनश्च ॥
S.S.Utt. 44/13

Complications of *Pāṇḍu* and *Kāmala* are anorexia, thirst, vomiting, fever, headache, weakness of digestive fire, swelling of the throat, debility, fainting, exhaustion and discomfort in cardiac region.

उपशय/पथ्य (Upaśaya/Pathya)

कोष्ठाश्रय कामला (Koṣṭhāśraya Kāmalā)

Like *Pāṇḍuroga*

शाखाश्रय कामला (Śākhāśraya Kāmalā)

बर्हितित्तिरिदक्षाणां रुक्षाम्लैः कटुकै रसैः ॥
शुष्कमूलककौलत्थैर्युषैश्चान्नानि भोजयेत् ।
मातुलुङ्गरसं क्षौद्रपिप्पलीमरिचान्वितम् ॥
सनागरं पिबेत् पित्तं तथाऽस्यैति स्वमाशयम् ।
C.S.Ci. 16/128-130

Patients of *Śākhāśraya Kāmalā* should consume food with the soup of the meat of peacock, partridge and cock, intake of ununctous, sour and pungent substances; vegetable soup prepared from dry radish and *Kulattha*; juice of *Mātuluṅga,* mixed with honey, *Pippali*, *Marica* and *Śuṇṭhi.* The above substances will help to bring the *Pitta* to its normal place.

Halīmaka (C.S.Ci. 16/134-137)

- Recipe prepared from buffalo milk by adding the juice of *Guḍūcī* and milk.
- After oleation, *Trivṛt* mixed with *Āmalaki* juice should be given to induce purgation. *Drakṣāvalehya, Dhātryāvalehya.*

Different varieites of *Yāpana basti, Kṣīra basti* and *Anuvāsana basti.*

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Koṣṭhāśraya Kāmala Patients should not take *Pitta* aggravating factors. *Śākhāśraya Kāmala* patients should not consume *Pitta* alleviating factors until the stool of the patient acquires the colour of *Pitta.* All the causative factors.

MCQs

1. *Dosha* predominance *Haleemaka*
   a. *Kapha-Pitta*　b. *Tridoshaja*
   c. *Vata-Pitta*　d. *Paittika*

2. *Kaphasammur chitovayuhu Sthanatpittamksipedbalee*-such pathogenetic events described in context to
   - a. *Shakhashraya kamala*
   - b. *Kumbha kamala*
   - c. *Shvasaroga*
   - d. *Vatarakta*
3. Prognosis of *Kumbha kamala*
   - a. *Sadhya* b. *Krichram*
   - c. *Yapya* d. *Asadhya*
4. *Streeshuaharshomridujwara* is seen in
   - a. *Kaphajapandu* b. *Kumbha kamala*
   - c. *Haleemaka* d. *Kamala*
5. भेकाभोदुर्बलेन्द्रिय is the feature of
   - a. *Kamala* b. *Pandu*
   - c. *Sopha* d. *Haleemaka*
6. Which among the following is not the feature of *Haleemaka*
   - a. *Streeshuaharsham* b. *Balautsahakshaya*
   - c. *Mridujwara* d. *Bhinnavit*
7. *Panaki* disease is mentioned by
   - a. *Charaka* b. *Bhavaprakasa*
   - c. *Madavanidana* d. *Susruta*
8. सन्तापोभिन्नवर्चस्त्वंबहिरन्तश्चपीतता पाण्डुतानेत्रयोर्यस्य ______
   - a. *Kamala* b. *Panaki*
   - c. *Haleemaka*
   - d. *Mrtbhakshanajanyapandu*
9. कालान्तरात्खरीभूताकृच्छ्रास्यात् ____________
   - a. *Haleemaka*
   - b. *Mrtbhakshanajanyapandu*
   - c. *Kumbha kamala*
   - d. *Panaki*
10. Which among the following is not the *asadyalakshana* of *Kamala*
    - a. *Krishna peetasakrit and mutra*
    - b. *Raktachardhi*
    - c. *Nashtasamjna*
    - d. *Bhinnavarchas*

Answer

1.	c	2.	a	3.	b
4.	c	5.	a	6.	d
7.	c	8.	b	9.	c
10.	d				

CHAPTER

27

Udara Roga

Introduction

Diseases of the abdomen. Long standing *mandāgni* (diminished digestive power) and *ajīrṇa* (indigestion) cause generalized enlargement of the abdomen and this indisposition is identified as *Udara Roga*. Out of the eight types of *Udara roga* four are *Doṣaja* (generated by the vitiated humors) viz. *Vātodara*, *Pittodara*, *Kaphodara* and *Sannipātodara*, the fifth is due to the enlargement of spleen or liver i. e. *Plīhodara* and *Yakṛddālyudara*, the sixth is due to the intestinal obstruction i. e. *Baddha-Gudodara*, the seventh is due to the injury of the abdomen i. e. *Parisrāvyudara* and while the last of the *Udararoga* is due to the accumulation of fluid i. e. *Jalodara*.

हेतु (Hetu)

अत्युष्णलवणक्षारविदाह्यम्लगराशनात् ।
मिथ्यासंसर्जनाद्रूक्षविरुद्धाशुचिभोजनात् ॥
प्लीहार्शोग्रहणीदोषकर्शनात् कर्मविभ्रमात् ।
क्लिष्टानामप्रतीकाराद्रौक्ष्याद्वेगविधारणात् ॥
स्त्रोतसां दूषणादामात् संक्षोभादतिपूरणात् ।
अर्शोबालशकृद्रोधादन्त्रस्फुटनभेदनात् ॥
अतिसंचितदोषाणां पापं कर्म च कुर्वताम् ।
उदराण्युपजायन्ते मन्दाग्नीनां विशेषतः ॥

C.S.Ci. 13/12-15

Etiological factors of *Udara roga* are as follows :- Excessive intake of hot, saline, alkaline, burning, sour and poisoned food; improper dietetic regimen after evacuative procedures, intake of rough, antagonistic and unclean food, emaciation due to splenomegaly, piles and *Grahaṇī roga*. Improper administration of evacuative therapy and neglecting the treatment of these disorders i:e piles, splenomegaly etc. roughness, suppression of natural urges and morbidity in channels. *Āmadoṣa*, intake of irritating foods and drinks, over saturation, obstruction due to piles, hairs, stool, perforation and tearing of intestines, excessive accumulation of vitiated *Doṣās* and indulgence in sinful acts.

The places of eastern country are known as sweet, cold and heavy. These are first of all *Kumāravartani*, then *Kaṭivarṣa* in *Māgadha*, the *Mahārāṣṭra* and also *Ṛṣabhadvīpa*, *Paunḍravardhanaka* and also *Mrittika*, *Vardhamānaka, Karvaṭa, Mātaṅga, Tāmralipta, Ciraka, Priyaṅgu, Kauśalya, Kaliṅga* and *Priṣṭapūraka.* The mortals are susceptible to – *Plīha (Splenomegaly) and Galagaṇḍa (Goitre). (K.S.Khi. 25/7-11.1)*

- All diseases developed due to heat of the body and psyche of the God and sages running due to fear of being killed in *Yajña* of *Dakṣa*. (K.S.K. 9/14)
- Due to swimming and floating, the *Plīha roga* manifests. (K.S.K. 9/15)

Complication of non-observance of phased diet after cleansing therapy leads to the manifestation of many complications like *Udara* (K.S.Su. 24/16-17)

पूर्वरूप (Pūrvarūpa)

क्षुन्नाशः स्वाद्वतिस्निग्धगुर्वन्नं पच्यते चिरात् ।
भुक्तं विदह्यते सर्वं जीर्णाजीर्णं न वेत्ति च ॥
सहते नातिसौहित्यमीषच्छोफश्च पादयोः ।
शश्वद्बलक्षयोऽल्पेऽपि व्यायामे श्वासमृच्छति ॥
वृद्धिः पुरीषनिचयो रूक्षोदावर्तहेतुका ।
बस्तिसन्धौ रुगाध्मानं वर्धते पाठ्यतेऽपि च ॥
आतन्यते च जठरमपि लघ्वल्पभोजनात् ।
राजीजन्म वलीनाश इति लिङ्गं भविष्यताम् ॥

C.S.Ci. 13/16-19

Lack of appetite; delayed digestion of sweet, too unctous and heavy food; burning of the entire food taken (with hyperacidity); absence of feeling of digestion or indigestion; intolerance to over eating; slight oedema on feet; continuous deterioration of strength; dyspnoea even on slight physical exertion; abdominal enlargement; accumulation of faeces; pain in pelvic region due to roughness and upward movement of *Vāta*; increase of tympanitis; increasing, tearing and expansion of abdomen even on light and little food; appearance of streaks; disappearance of abdominal wrinkles; loss of strength, colour, complexion & desire for food; appearance of lines over abdomen, and also folds of abdomen; uncertain understanding of digestion and burning sensation during digestion.

उदररोग संप्राप्ति (Saṁprāpti *of* Udara Roga)

अग्निदोषान्मनुष्याणां रोगसङ्घाः पृथग्विधाः।
मलवृद्ध्या प्रवर्तन्ते विशेषेणोदराणि तु॥
मन्देऽग्नौ मलिनैर्भुक्तैरपाकाद्दोषसंचयः।
प्राणाग्न्यपानान् संदूष्य मार्गान्रुद्ध्वाऽधरोत्तरान्॥
त्वङ्मांसान्तरमागम्य कुक्षिमाध्मापयन् भृशम्।
जनयत्युदरं तस्य हेतुं शृणु सलक्षणम्॥
C.S.Ci. 13/9-11

सर्वमेवोदरं प्रायो दोषसङ्घातजं मातम्॥ *C.S.Ci. 13/95*

All varieties of *Udara roga* are generally caused by the congregation of *Doṣās*. In human beings different disease groups particularly the abdominal diseases arise from the defect of *Agni* due to increase of *Malās*. If person indulges in foods and activities during weak digestion leads to improper digestion and gradually accumulation of *Doṣās*, which affecting *Prāṇa, Agni* and *Apāna,* and blocking upward and downward passages reaches the interspace between skin and muscle and blows up the abdomen.

रुद्ध्वा स्वेदाम्बुवाहीनि दोषाः स्रोतांसि संचिताः।
प्राणाग्न्यपानान् संदूष्य जनयन्त्युदरं नृणाम्॥
C.S.Ci. 13/20

Accumulated *Doṣās* obstruct the *Svedavaha* & *Ambuvaha Srotas* and vitiate *Prāṇa, Agni* and *Apāna* leading to the manifestation of *Udara roga.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi doṣās*
↓
Disturbance of *Agni*
↓
If person indulges in foods and activities during weak digestion leads to
improper digestion and gradually accumulation of *Āma* and other *Doṣās*
↓
Congregation of *Vatādi doṣās* and *Rasādi dhātus* in the *Annavaha Srotas*
↓
Which affects the *Prāṇa, Agni* and *Apāna*
↓
Leading to blockage of upward and downward passages
↓
Favours the accumulation of *doṣās* in the interspace between skin and
muscle in the *Udarapradeśa* (abdomen)
↓
Distension of abdomen
↓
Udara Roga

उदररोग संप्राप्ति घटक (Samprāpti Ghaṭaka *of* Udara Roga)

Components of *Samprāpti* are as follows-

Doṣa : *Tridoṣaja*
Vāta → *Prāṇa, Apāna, Samāna*
Pitta → *Pācaka*
Kapha → *Kledaka*
Dūṣya : *Dhātu* → *Rasa, Rakta, Māmsa*
Upadhātu → *Tvacā, Vasā*
Śārīrika Mala → *Mūtra, Puriṣa*
Dhātu Mala → *Sveda*
Agni : *Jaṭharāgni, Dhātvāgni*
Agniduṣṭi : *Mandatā*
Āma : *Āgnijanya*

Srotas : *Svedavaha, Ambuvaha, Rasavaha, Raktavaha, Māmsavaha, Annavaha,*
Srotoduṣṭi : *Saṅga, Vimārgagamana*
Udbhavasthāna : *Āmāśayottha, Koṣṭha*
Adhiṣṭhāna : *Udara, Tvacā (Tvak Māmsantaram)*
Vyaktasthāna : *Udara, Yakṛt, Plīhā*
Rogamārga : *Abhyantara*
Sancārasthāna : *Sirāḥ (Su. Ni 7/6), Rasāyani*
Svabhāva : *Cirakāri*
Prabhāva : *Vātodara, Pittodara, Kaphodara, Plīhodara, Sannipātodara & Udakodara,* the subseqet ones are more and more difficult for treatment than the previous ones.
Roga bheda : *8 types*

सामान्य लक्षण (Sāmānya Lakṣaṇa)

General Symptoms of abdominal diseases

कुक्षेराध्मानमाटोपः शोफः पादकरस्य च ।
मन्दोऽग्निः श्लक्ष्णगण्डत्वं कार्श्यं चोदरलक्षणम् ॥
C.S.Ci. 13/21

आध्मानं गमनेऽशक्तिर्दौर्बल्यं दुर्बलाग्निता ।
शोफः सदनमङ्गानां सङ्गो वातपुरीषयोः ।
दाहस्तृष्णा च सर्वेषु जठरेषु भवन्ति हि ॥
S.S. Ni. 7/24-25

Tympanitis and gurgling sound in abdomen; oedema on hands and feet; poor digestion; smooth cheek; emaciation; flatulence; inability to walk; debility; swelling & weakness of body parts; obstruction of flatus & stool; burning sensations and thirst.

Classification of Udara Roga

पृथग्दोषैः समस्तैश्च प्लीहबद्धक्षतोदकैः ।
संभवन्त्युदराण्यष्टौ तेषां लिङ्गं पृथक् शृणु ॥
C.S.Ci. 13/22

8 types

1. *Vātaja Udara roga*
2. *Pittaja Udara roga*
3. *Kaphaja Udara roga*
4. *Sannipātaja Udara roga*
5. *Plīhodara roga*
6. *Baddhodara roga*
7. *Kṣatodara roga*
8. *Udakodara roga*

Five types of *Plīha Doṣa* – *Vātaja, Pittaja, Kaphaja, Sannipātaja* and *Raktaja*. (C. Su. 19/3).

वातज उदर रोग हेतु एवं संप्राप्ति (Vātaja Udara Roga Hetu evam Samprāpti)

रूक्षाल्पभोजनायासवेगोदावर्तकर्शनैः ।
वायुः प्रकुपितः कुक्षिहृद्बस्तिगुदमार्गगः ॥
हत्वाऽग्निं कफमुद्धूय तेन रुद्धगतिस्ततः ।
आचिनोत्युदरं जन्तोस्त्वङ्मांसान्तरमाश्रितः ॥
C.S.Ci. 13/23-24

Etiological factors and pathogenesis of *Vātaja Udara Roga* are as follows : *Vāta* gets aggravated due to intake of rough food, less consumption of food, exertion, suppression of natural urges, *Udāvarta* and emaciation.

Aggravated *Vāta* reaches belly, cardiac region, urinary bladder and anus, suppresses the power of digestion & stimulates *Kapha*. This *Kapha* obstructs the movement of *Vāta* and thus enlarges the abdomen by residing in the interspace between skin & muscle.

वातज उदर रोग लक्षण (Vātaja Udara Roga Lakṣaṇa)

तस्य रूपाणि–कुक्षिपाणिपादवृषणश्वयथुः, उदरविपाटनम्, अनियतौ च वृद्धिह्रासौ, कुक्षिपार्श्वशूलोदावर्ताङ्गमर्दपर्वभेदशुष्ककासकार्श्यदौर्बल्यारोचकाविपाकाः, अधोगुरुत्वं, वातवर्चोमूत्रसङ्ग, श्यावारुणत्वं च नखनयनवदनत्वङ्गमूत्रवर्चसाम्, अपि चोदरं तन्वसितराजीसिरासंततम् आहंतमाध्मातदृतिशब्दवद्भवति, वायुश्चोर्ध्वमधस्तिर्यक् च सशूलशब्दश्चरति, एतद्वातोदरमिति विद्यात् ॥ *C.S.Ci. 13/25*

Signs & symptoms of *Vātaja Udara Roga* are as follows : Swelling in the belly, hands, feet and scroatum; tearing of abdomen; increase & decrease of the swelling in the abdomen without any apparent cause; colic pain in the belly & sides; *Udāvarta;* general malaise, pain in small joints; dry cough; emaciation; weakness; anorexia; indigestion; heaviness in the lower abdomen; non elimination of flatus, stool & urine; appearance of blackish or reddish tinge in nails, eyes, face, skin, urine & stool; appearance of thin & black net work of veins over the abdomen; on percussion, sounds like the beat of an inflated leather bag and movement of *Vāta* upwards, downwards & sidewards alongwith colic pain & sound.

पित्तज उदर रोग हेतु एवं संप्राप्ति (Pittaja Udara Roga Hetu evam Samprāpti)

कट्म्ललवणात्युष्णातीक्ष्णाग्न्यातपसेवनैः ।
विदाह्यध्यशनाजीर्णैश्चाशु पित्तं समाचितम् ॥
प्राप्यानिलकफौ रुद्ध्वा मार्गन्मार्गमास्थितम् ।
निहन्त्यामाशये वह्निं जनयत्युदरं ततः ॥
C.S.Ci. 13/26-27

Etiological factors & pathogenesis of *Pittaja Udara Roga* are as follows : *Pitta* accumulates due to intake of pungent, sour, saline & very hot food items; exposure to intense fire & sun; *Vidāhi* food and intake of food before the digestion of previous meals.

Aggravated *Pitta* joins with *Vāta* & *Kapha* obstruct the passage and moving upwards it suppresses the *Agni* in the stomach and manifest *Pittaja Udara Roga.*

पित्तज उदर रोग लक्षण (Pittaja Udararoga Lakṣaṇa)

तस्य रूपाणि–दाहज्वरतृष्णामूर्च्छातीसारभ्रमाः, कटुकास्यत्वं, हरितहारिद्रत्वं नखनयनवदनत्वङ्मूत्रवर्चसाम्, अपि चोदरं नीलपीतहारिद्रहरिताम्रराजीसिरावनद्धं, दह्यते, दूयते, धूप्यते, ऊष्मायते, स्विद्यते, क्लिद्यते, मृदुस्पर्शं क्षिप्रपाकं च भवति; एतत् पित्तोदरमिति विद्यात्। *C.S.Ci. 13/28*

Signs & symptoms of *Pittaja Udara Roga* are as follows : Burning sensation, fever, thirst, fainting, diarrhoea, giddiness, pungent taste in the mouth, appearance of green & yellow tinge in nails, eyes, face, skin, urine & stool; prominence of blue, yellow, deep yellow, green or coppery streaks & veins on abdomen which has burning sensation, pain, fuming, heating, sweating, moistening & is soft on palpation & prone to immediate inflammation.

कफज उदर रोग हेतु एवं संप्राप्ति (Kaphaja Udararoga Hetu evam Samprāpti)

अव्यायामदिवास्वप्नस्वाद्वतिस्निग्धपिच्छिलैः ।
दधिदुग्धौदकानूपमांसैश्चाप्यतिसेवितैः ॥
क्रुद्धेन श्लेष्मणा स्रोतःस्वावृतेष्वावृतोऽनिलः ।
तमेव पीडयन् कुर्यादुदरं बहिरन्त्रगः ॥
C.S.Ci. 13/29-30

Kapha accumulates due to lack of exercise, day sleep; intake of sweet, unctous and slimy food; curd; milk; meat of aquatic & marshy animals.

Aggravated *Kapha* obstruct the channels of circulation as a result of which *Vāta* having been blocked in its passage comes out of intestine & produces *Kaphaja Udara Roga.*

कफज उदर रोग लक्षण (Kaphaja Udara Roga Lakṣaṇa)

तस्य रूपाणि-गौरवारोचकाविपाकाङ्गमर्दाः, सुप्तिः, पाणिपादमुष्कोरुशोफः, उत्क्लेशनिद्राकासश्वासाः, शुक्लत्वं च नखनयनवदनत्वङ्मूत्रवर्चसाम्ः अपि चोदरं शुक्लराजीसिरासंततं, गुरु, स्तिमितं, स्थिरं, कठिनं च भवति; एतच्छलेष्मोदरमिति विद्यात् ॥ *C.S.Ci. 13/31*

Signs & symptoms of *Kaphaja Udara Roga* are as follows:– Heaviness, anorexia, indigestion & malaise, numbness, appearance of swelling in hands, legs, scrotum and thighs, nausea, excessive sleep, cough, dyspnoea, whiteness of nails, eyes, face, skin, urine & stool, prominence of white streaks & veins all over the abdomen and abdomen becomes heavy, timid, immobile & hard.

सन्निपातज उदर रोग हेतु एवं संप्राप्ति (Sannipātaja Udara Roga Hetu evam Samprāpti)

दुर्बलाग्नेरपथ्यामबिरोधिगुरुभोजनैः ।
स्त्रीदत्तैश्च रजोरोमविण्मूत्रास्थिनखादिभिः ॥
विषैश्च मन्दैर्वाताद्याः कुपिताः संचयं त्रयः ।
शनैः कोष्ठे प्रकुर्वन्तो जनयन्त्युदरं नृणाम् ॥
C.S.Ci. 13/32-33

Simultaneous acumulation of all the *Doṣās* due to intake of unwholesome, uncooked, mutually contradictory & heavy food during weak power of digestion; when unwholesome ingredients like menstrual blood, hair, stool, urine, bone, nails etc. are administered by a woman and slow poisoning (*dūṣīviṣa*). All the three *Doṣās* gets aggravated & accumulates gradually in the abdomen & manifest *Sannipātaja Udara Roga.*

सन्निपातज उदर रोग लक्षण (Sannipātaja Udara Roga Lakṣaṇa)

Signs & symptoms of *Sannipātaja Udara Roga* are as follows : Appearance of signs & symptoms of all the three *Doṣās;* morbidity of nails etc. with all the types of colours; appearance of network of veins over the abdomen having various colours and streaks in the abdomen.

सन्निपातज उदर (दूष्योदर) [Sannipātaja Udara (Dūṣyodara)]

स्त्रियोऽन्नपानं नखरोममूत्रविडार्त्तवैर्युक्तमसाधुवृत्ताः ।
यस्मै प्रयच्छन्त्यरयो गरांश्च दुष्टाम्बुदूषीविषसेवनाद्वा ॥
तेनाशु रक्तं कुपिताश्च दोषाः कुर्वन्ति घोरं जठरं त्रिलिङ्गम् ।
तच्छीतवाताभ्रसमुद्भवेषु विशेषतः कुप्यति दह्यते च ॥
स चातुरो मूर्च्छति सम्प्रसक्तं पाण्डुः कृशः शुष्यति तृष्णया च ।
प्रकीर्त्तितं दूष्युदरं तु घोरं ॥

S. S.Ni. 7/11-14

Uncultured women serving food mixed with nails, hairs, urine, excreta, menstrual blood etc. or enemies administering artificial poisons or use of contaminated water, impoverished poisons etc. leading to aggravation of all the three *Doṣās* along with *Rakta* & manifest enlargement of *Udara* accompanied with symptoms of all the three *Doṣās.* This disease becomes excacerbated espescially during the time of cold breeze & cloudy sky, burning sensation, fainting, anaemia, emaciation & thirst. This condition is also called *Dūṣyodara* & it is dreadful.

प्लीहोदर एवं यकृद्दाल्युदर हेतु एवं सम्प्राप्ति (Plīhodara evam Yakṛddālyudara Hetu evam Samprāpti)

रसप्रदोषजारोगाः वक्ष्यन्ते रक्तदोषजाः।
कुष्ठवीसर्पपिडका रक्तपित्तमसृद्गरः॥
गुदमेढ्रास्यपाकश्च प्लीहा गुल्मोऽथ विद्रधिः।
नीलिका कामला व्यङ्गः पिप्लवस्तिलकालकाः॥

C.S.Su. 28/11-12

अशितस्यातिसंक्षोभाद्यानयानातिचेष्टितैः ।
अतिव्यवायभाराध्ववमनव्याधिकर्शनैः ॥

C.S.Ci. 13/36

It manifests due to affliction of *rakta*; excessive jerking due to travelling by vehicle; excessive movements; excessive sexual intercourse; excessive load carrying; excessive travelling on foot; excessive vomitting and debility caused by some diseases.

संप्राप्ति (Samprāpti)

वामपार्श्वाश्रितः प्लीहा च्युतः स्थानात् प्रवर्धते ।
शोणितं वा रसादिभ्यो विवृद्धं तं विवर्धयेत् ॥

C.S.Ci. 13/36

तस्य प्लीहा कठिनोऽष्ठीलेवादौ वर्धमानः कच्छपसंस्थान उपलभ्यतेः स चोपेक्षितः क्रमेण कुक्षिं जठरमग्न्यधिष्ठानं च परिक्षिपन्नुदरमभिनिर्वर्तयति ॥ *C.S.Ci. 13/37*

Due to the above mentioned etiological factors the spleen located in the left side gets displaced from its normal place and thus become enlarged or the blood increased (accumulated) through *Rasa* etc. also enlarge it.

The spleen becomes hard like a small stony piece in the beginning gradually increases and become like a tortoise. If neglected, it gradually overlaps belly, abdomen and the seat of digestive fire.

लक्षण (Lakṣaṇa)

तस्य रूपाणिदौर्बल्यारोचकाविपाकवर्चोमूत्रग्रहतमःप्रवेश–पिपासाङ्ग मर्दच्छर्दिमूर्च्छाङ्गसादकासश्वासमृदुज्वरानाहाग्नि–नाशकार्श्यास्यवैरस्यपर्वभेदकोष्ठवातशूलानि, अपि चोदरमरुणवर्णं विवर्णं वा नीलहरितहारिद्रराजिमद्भवति; एवमेव यकृदपि दक्षिणपार्श्वस्थं कुर्यात, तुल्यहेतुलिङ्गौषधत्वात्तरस्य प्लीहजठर एवावरोध इति; एतत् प्लीहोदरमिति विद्यात् ॥

C.S.Ci. 13/37-38

Debility, anorexia, indigestion, retention of stool and urine, unconsciousness, thirst, body-ache, vomiting, fainting, lassitude, cough, dyspnoea, mild fever, hardness in bowels, loss of fire, emaciation, tastelessness in mouth, tearing pain in smaller joints, gas formation, pain in belly, reddish or abnormal tinge or appearance of blue, green or yellow streaks on abdomen.

In the same way liver becomes enlarged on right side of the abdomen with above symptoms is known as *Yakṛddhā lyodara.*

Classification of Plīhodara

Vātaja Plīhodara – Upward movement of *Vayu,* pain and hardness in bowels.

Pittaja Plīhodara – Burning sensation, mental confusion, thirst and fever.

Kaphaja Plīhodara– Heaviness, anorexia and hardness.

Raktaja Plīhodara – Burning sensation, thirst, tastelessness, heaviness in the body and fainting (*Caraka Cakrapāṇi).*

बद्धगुदोदर हेतु एवं संप्राप्ति (Baddhagudodara Hetu evam Samprāpti)

पक्ष्मबालैः सहान्नेन भुक्तैर्बद्धायने गुदे ।
उदावर्तैस्तथाऽर्शोभिरन्त्रसंमूर्च्छनेन वा ॥
अपानो मार्गसंरोधाद्धत्वाऽग्निं कुपितोऽनिलः ।
वर्चःपित्तकफान् रुद्ध्वा जनयत्युदरं ततः ॥
C.S.Ci. 13/39-40

Vāta gets aggravates due to obstruction in anal passage caused by intake of eyelashes, hairs etc. with meals, *Udāvarta,* piles, *Āntrasammūrchana* (intussusception), obstruction to the passage of *Apānavāta.*

Aggravated *Apānavāta,* suppresses *Agni* & obstruct the movement of stool, *Pitta* & *Kapha* as a result of which *Baddhagudodara* manifest.

बद्धगुदोदर लक्षण (Baddhagudodara Lakṣaṇa)

तस्य रूपाणितृष्णादाहज्वरमुखतालुशोषोरुसादकासश्वास–दौर्बल्यारोचकाविपाकवर्चोमूत्रसङ्गाध्मानच्छर्दिक्षवथुशिरो–हृन्नाभिगुदशूलानि, अपि चोदरं मूढवातं स्थिरमरुणं नीलराजि सिरावनद्धराजिकं वा प्रायो नाभ्युपरि गोपुच्छवदभिनिर्वर्तत इति; एतद्बद्धगुदोदरमिति विद्यात् ॥ *C.S.Ci. 13/41*

Signs & symptoms of *Baddhagudodara* are as follows : Morbid thirst, burning sensation, fever, dryness of mouth & palate, prostration in the thighs, cough, dyspnoea, weakness, anorexia, indigestion, retention of stool & urine, abdominal distension, vomiting, sneezing, haedache, & colic pain in the regions of the heart, umbilicus and anus; absence of peristaltic movement in the abdomen; abdomen becomes confounded by *Vāta,* stable, reddish & blue net work of veins and streaks and appearance of elongated swelling like the shape of the tail of the cow over the umbilical region.

छिद्रोदर हेतु एवं संप्राप्ति (Chidrodara Hetu evam Samprāpti)

शर्करातृणकाष्ठास्थिकण्टकैरन्नसंयुतैः ।
भिद्येतान्त्रं यदा भुक्तैर्जृम्भयाऽत्यशनेन वा ॥
पाकं गच्छेद्रसस्तेभ्यश्छिद्रेभ्यः प्रस्रवद्बहिः ।
पूरयन् गुदमन्त्रं च जनयत्युदरं ततः ॥
C.S.Ci. 13/42-43

Etiological factors & pathogenesis of *Chidrodara* are as follows : Perforation of intestines due to ingestion of gravels, grass, wooden or bony piece or thorn with food; yawning, over eating.

Wound of the intestine gets suppurated and discharges leading to accumulation of secretion in the rectum and intestines and produces *Chidrodara*.

छिद्रोदर लक्षण (Chidrodara Lakṣaṇa)

तस्य रूपाणि–तदधो नाभ्याः प्रायोरभिवर्धमानमुदकोदरं भवति, यथाबलं य दोषाणां रूपाणि दर्शयति, अति चातुरःसलोहितनील–पीतपिच्छिलकुणपगन्ध्यामवर्चं उपवेशते, हिक्काश्वासकास–तृष्णाप्रमेहारोचकाविपाकदौर्बल्यपरीतश्च भवति, एतच्छिद्रोदरमिति विद्यात् ॥ *C.S.Ci. 13/44*

Signs & symptoms of *Chidrodara* are as follows : Abdomen gets distended below the umbilical region; it subsequently grows to cause *Udakodara,* exhibits the symptoms of *Doṣās* according to their dominancy; patient passes stool which is either red, blue, yellow, slimy or having the odour of a dead body, *Āma,* hiccup, dyspnoea, cough, thirst, polyuria, anorexia, indigestion and debility.

Chidrodara is termed as *Parisrāvyudara* or *Āgantuja Udara* by *Suśruta.*

उदकोदर हेतु एवं संप्राप्ति (Udakodara Hetu evam Samprāpti)

स्नेहपीतस्य मन्दाग्नेः क्षीणस्यातिकृशस्य वा अत्यम्बुपाना–न्नष्टेऽग्नौ मारुतः क्लोम्नि संस्थितः ॥ स्रोतःसु रुद्धमार्गेषु

कफश्चोदकमूर्च्छितः । वर्धयेतां तदेवाम्बु स्वस्थानादुदराय तौ ॥
C.S.Ci. 13/45-46

Agni becomes weak because of excessive intake of water after the administration of oleation therapy or by a person suffering from *Mandāgni* or by an individual who is cachectic or excessively emaciated. As a result of this, *Vāta* located in *Kloma* gets interrupted with *Kapha* and *Udaka Dhātu* increases the quantity of that water in the obstructed channels of circulation leading to manifestion of *Udakodara.*

उदकोदर लक्षण (Udakodara Lakṣaṇa)

तस्य रूपाणिअनन्नकाङ्क्षापिपासागुदस्त्रावशूलश्वास–कासदौर्बल्यानि, अपि चोदरं नानावर्णराजिसिरासंततमुदक–पूर्णदृतिक्षोभसंस्पर्शं भवति, एतदुदकोदरमिति विद्यात् ॥
C.S.Ci. 13/47

Signs & symptoms of *Udakodara* are as follows : Loss of appetite, morbid thirst, discharge from the anus, colic pain, dyspnoea, cough, general debility, appearance of network of veins having different colours over the abdomen and on palpation has fluctuation like leather bag filled with water. It is known as *Dakodara.*

उदकोदर साध्यासाध्यता (Udakodara Sādhyāsādhyatā)

तत्र अचिरोत्पन्नमनुपद्रवमनुदकप्राप्तमुदरं त्वरमाणश्चिकित्सेत्; उपेक्षितानां ह्येषां दोषाः स्वस्थानादपवृत्ता परीपाकाद्द्रवीभूताः सन्धीन् स्त्रोतांसि चोपक्लेदयन्ति, स्वेदश्च वाह्येषु स्रोतःसु प्रतिहतगतिस्तिर्यगवतिष्ठमानस्तदेवोदकमाप्याययतिः तत्र पिच्छोत्पत्तौ मण्डलमुदरं गुरु स्तिमितमाकोठितमशव्दं मृदुस्पर्शमपगत–राजीकमाक्रान्तं नाभ्यामेवोपसर्पति । ततोऽनन्तरमुदकप्रादुर्भावः । तस्य रूपाणिकुक्षेरतिमात्रवृद्धिः सिरान्तर्धानगमनम्, उदकपूर्णदृति–संक्षोभसंस्पंशत्वं च ॥ *C.S.Ci. 13/48*

Prognosis of *Udara Roga* are as follows : Physician should treat the condition immediately before the appearance of any complications & before accumulation of liquid in the abdomen. If the treatment is neglected, then the vitiated *Doṣās* get displaced & become liquified as a result of *Paripāka,* cause stickiness in the joints & channels of circulation and divert sweat from the external channels as a result of which it moves sideways, this zigzag moving sweat adds to the quantity of water already accumulated on the abdomen. The appearance of this sticky liquid makes the abdomen round, heavy and numb. The abdomen becomes dull in percussion & soft to touch. Thereafter, the net work of veins disappears. During this period, the umbilical region is primarily afflicted and from these the disease spreads in the remaining parts of the abdomen. Thereafter water starts accumulating in the abdomen leading to excessive enlargement of the sides of the abdomen, disappearance of the network of veins, on palpation physician feels as if the abdomen is a leather sack filled with water.

Udakodara has two stages. During the first stage, water accumulation does not takes place in the abdomen & it is called *'Ajātodaka'* stage. When the vitiated *Doṣās* get matured they become liquefied by nature. Because of the obstruction in the channels of circulation in the exterior of the body, the sweat becomes incapable of going out as it happens in normal conditions. If moves inwards as a result of which sticky material appears in the abdomen. According to some scholars, *picchā* is like the scum which comes out while boiling rice. It is because of the accumulation of this sticky liquid inside the muscle tissues, that the physician by palpation feels the abdomen like a leather sack filled with water (C.S.Ci 13/48, *Cakrapāṇi*)

अजातोदक उदर (Ajatodaka Udara)

अजातशोथमरुणं सशब्दं नातिभारिकम् ।
सदा गुडगुडायच्च सिराजालगवाक्षितम् ॥
नाभिं विष्टभ्य वायुस्तु वेगं कृत्वा प्रणश्यति ।
हृन्नाभिवङ्क्षणकटीगुदप्रत्येकशूलिनः ॥
कर्कशं सृजतो वातं नातिमन्दे च पावके ।
लालया विरसे चास्ये मूत्रेऽल्पे संहते विषि ॥
अजातोदकमित्येतैर्लिङ्गैर्विज्ञाय तत्त्वतः ।
उपाक्रमेद्भिषग्दोषबलकालविशेषवित् ॥
C.S.Ci. 13/55-58

Signs & symptoms of *Ajatodaka Udara* are as follows : Abdomen without swelling, reddish, resonant on percussion, not very heavy, always with gurgling sounds, full of veinous network, *Vāta* distending the navel & disappearing after impelling the urge; pain in the heart, navel, groin, waist & anus; passing hard flatus, digestive fire not very mild, tastelessness in mouth due to salivation, scanty urine, hard stool. It should be taken as that devoid of fluid & should be treated by the physician knowing the variation of *Doṣa*, strength & time.

उपद्रव (Upadrava)

तदाऽऽतुरमुपद्रवाः स्पृशन्तिछर्द्यतीसारतमकतृष्णाश्वास–कासहिक्कादौर्बल्यपाश्र्वशूलारुचिस्वरभेदमूत्रसङ्गादयः; तथाविध मचिकित्स्यं विद्यादिति ॥ *C.S.Ci. 13/49*

Complication of *Udara Roga* are as follows : Vomiting, diarrhoea, feeling of darkness, thirst, dyspnoea, cough, hiccup, debility, pain in sides, anorexia, hoarseness of voice, retention of urine etc. Such patient should be declared as incurable.

अन्ते सलिलभावं हि भजन्ते जठराणि तु।
सर्वाण्येव परीपाकात्तदा तानि विवर्जयेत्॥
S.S.Ni. 7/25

All varieties of *Udara Roga* attain the stage of accumulation of fluid at their end stage due to ripening; then they should be rejected for treatment.

शूनाक्षं कुटिलोपस्थमुपक्लिन्नुत्वचम्।
बलशोणितमांसाग्निपरिक्षीणं च वर्जयेत्॥
श्वयथुः सर्वमर्मोत्थः श्वासो हिक्काऽरुचिः सतृट्।
मूर्च्छा च्छर्दिरतीसारो निहन्त्युदरिणं नरम्॥
जन्मनैवोदरं सर्वं प्रायः कृच्छ्रतमं मतम्।
बलिनस्तदजाताम्बु यत्नसाध्यं नवोत्थितम्॥
C. Ci. 13/52-54

One should reject the patient having swollen eyes, crooked private parts, moistened thin skin, and loss of strength, blood, flesh and digestive power. Swelling in all the vital parts, dyspnoea, hiccup, anorexia, thirst, fainting, vomiting and diarrhoea lead to death of the patient. *Udara roga* as a whole is often the most difficult to be treated from the very start. However, it can be cured with effort if it is newly arisen, fluidless and patient is strong.

साध्यासाध्यता (Sādhyāsādhyatā)

अष्टावुदराणि पूर्वमुद्दिष्टानि। तेष्वसाध्यं बद्धगुदं परिस्त्रावि च; अवशिष्टानि कृच्छ्रसाध्यानि; सर्वाण्येव प्रत्याख्यायोपक्रमेत। तेष्वाद्यश्चतुर्वर्गो भेषजसाध्यः, उत्तरः शस्त्रसाध्यः, कालप्रकर्षात् सर्वाण्येव शस्त्रसाध्यानि वर्जयितव्यानि वा ॥ *S.S.Ci. 14/3*

Out of eight varieties of *Udara Roga Baddhagudodara* and *Parisrāvi Udara* are incurable and remaining are curable with difficulty; all are to be treated after informing the prognosis.

The first four i.e. *Vātaja, Pittaja, Kaphaja, Sannipātaja* are treatable with medicinal therapies; the remaining are treatable with surgical therapies; by lapse of time, all varieties becomes treatable by surgery or should be rejected for treatment.

वातात्पित्तात्कफात् प्लीह्नः सन्निपातात्तथोदकात्।
परं परं कृच्छ्रतरमुदरं भिषगादिशेत्॥
पक्षाद्बद्धगुदं तूर्ध्वं सर्वं जातोदकं तथा।
प्रायो भवत्यभावाय च्छिद्रान्त्रं चोदरं नृणाम्॥
C. Ci. 13/50-51

Udara roga caused by *Vāta, Pitta* and *Kapha, Plīha, Sannipāta* and *Udaka* should be known as of increasing severity in progressive order. After fort night, *baddhagudodara* becomes incurable. *Udakodara* (in its *jātodaka* stage) and *Chidrodara* are incurable right from the beginning.

उपशय/पथ्य (Upaśaya/Pathya)

औषध (Auṣadha)

Pañcakola Ghṛta, Nāgara Ghṛta, Citraka Ghṛta, Yavādya Ghṛta, Paṭolādya Cūrṇa, Nārāyaṇa Cūrṇa, Hapuṣādya Cūrṇa, Nilinyādya Cūrṇa, Snuhikṣīra Ghṛtam, Ṣaṭpala Ghṛta.

उदरी तु गुर्वभिष्यन्दिरूक्षविदाहिस्निग्धपिशितपरिषेकावगाहान् परिहरेत्; शालिषष्टिकयवगोधूमनीवारान् नित्यमश्नीयात् ॥
S.S.Ci. 14/4

Udara Roga patients should avoid foods which are hard for digestion, creates moisture inside the tissues, cuases dryness, produces burning sensation during digestion & fatty meat, taking bath & immershing in water. He should consume food prepared from *Śāli, Ṣaṣṭika, Yava, Godhūma* & *Nīvāra* daily. All *Udara Roga's* are caused by aggravated *Vāta* & by accumulation of *Malās* inside the body, experienced physicians praise the benefit of administering more of purgative therapy. (S.S.Ci. 14/9)

दोषैः कुक्षौ हि संपूर्णे वह्निर्मन्दत्वमुच्छति ॥
तस्माद्भोज्यानि भोज्यानि दीपनानि लघूनि च।
रक्तशालीन् यवान्मुद्गाञ्जाङ्गलांश्च मृगद्विजान्॥

पयोमूत्रासवारिष्टान्मधुसीधुं तथा सुराम्।
यवागूमोदनं वाऽपि यूषैरद्यादसैरपि ॥
मन्दाम्लस्नेहकटुभिः पञ्चमूलोपसाधितैः ।
औदकानूपजं मांसं शाकं पिष्टकृतं तिलान् ॥
व्यायामाध्वदिवास्वप्नं यानयानं च वर्जयेत्।
तथोष्णलवणाम्लानि विदाही निगुरूणि च ॥
नाद्यादन्नानि जठरी तोयपानं च वर्जयेत्।

C.S.Ci. 13/96-101

Udara is filled with vitiated *Doṣās*, the *Agni* gets diminished. Therefore, the patient should take such food which is light for digestion & which helps in the stimulation of digestive power. He should take red variety of *Śāli, Barley, Mudga*, meat of wild animals & birds, milk, cow's urine, *Āsava, Ariṣṭa*, honey, *Sidhu* & *Surā*. He may also consume *Yavāgu* & boiled rice prepared by boiling with decoction of *Pañcamūla* & added with slightly sour ingredients, unctous substances, like ghee & oil & pungent drugs along with vegetable soup or meat soup.

He should avoid aquatic & marshy meat & vegetables, flour preparations, sesamum seeds, physical exercise, travelling on food, day sleep & travelling by fast moving vehicles. Besides, he should avoid hot, salted, sour, burning & heavy foods, cereals & stricktly avoid taking water.

In *Udara Roga*, the abdomen gets completely afflicted with *Doṣas* as a result of which the power of digestion gets suppressed. Therefore, in such a condition, food ingredients, which stimulate the power of digestion and which are light for digestion, are given to the patient. The patient should take red variety of rice, barley, *Mudga*, meat of animals and birds inhabiting arid land, milk, cow's urine and *Āsava*, *Ariṣṭa*, *Madhu* as well as *Sidhu* types of alcoholic drink. (B.R. 40/2-3)

Diet and regimens for the patients of *Udara Roga*: Administration of the *Vamana* therapy, fasting, taking one year old pulse of *Kulattha*, old *Śāli* rice, barley, meat of the wild animals and birds, *Peya*, *Surā*, honey, liquor obtained from the plant *Madhuka* and *Sīdhu*, butter-milk, garlic pearls, *Eraṇḍa* oil, ginger, the *Śālinca* leaves, *Paṭola*, *Kārvellaka*, *Punarnavā*, fruits of plant *Śigru*, *Harada*, betal leaves, smaller cardamom, *Yavakṣāra*, *Lauha Bhasma*, milk and urine of the goat. Cow and buffalo, easily digestible and bitter items. Similarly, placing and roping the medicated cloth over the abdomen, cauterization, and the like are useful for the patient. In the case of the *Plīhodara*, the blood letting could be done through cutting the front portion of the left arm. In cases of *Baddhodara*, *Jalodara* and *Kṣatodara*, the the accumulated water, etc, could be removed out through cutting the portion below the naval. In cases of *Vātodara* the patient should be subjected to the smoke-therapy. This should be followed by the *Abhyañjana* therapy and finally by the *Anuvāsana* type of *Basti* therapy. Thus, in the cases of the *Udara* diseases, the regulation should be followed by considering the vitiated *Doṣa* in question. (B.R. 40/157-161)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Prohibitions of food and habits for the patients of *Udara Roga :* following measures are prohibited while suffering from problems of gastrointestinal tract-ointmenting, smoking, drinking water (in large quantity), blood letting, vomiting, riding, sleeping during day hours, taking hard exercises and the like. Similarly, following eatables should be avoided by the patient: eatables that are deeply fried into oils, meat of water born creatures and animals and birds of marshy region, leafy vegetables, sesame, hot and salty edibles, *Mudga* beans, incompatible meals, contaminated water, water of rivers originating in *Mahendrāchala* (probably the Himalayas) and the like. While suffering from constipation, *Svedana* therapy and taking meals that develop constipation should be avoided. The physician who is taking up the cases of problem of gastrointestinal tract should specially instruct the patient about these regulations. If he does not do so, he may lose his reputation. (B.R. 40/162-165)

The measures and products that have been considered as beneficial and harmful in the treatment of problems of gastrointestinal tract could be applied in the treatment of both spleen and liver problems. (B.R. 41/239).

Differential diagnosis of Doṣaja Udara

S.No.	*Vātodara*	*Pittodara*	*Kaphodara*	*Sannipātodara*
Etiology	*Vāta* gets aggravated due to intake of rough food, less consumption of food, exertion, suppression of natural urges, *Udāvarta* and emaciation	*Pitta* accumulates due to intake of pungent, sour, saline & very hot food items; exposure to intense fire & sun; *Vidahi* food and intake of food before the digestion of previous meals	*Kapha* accumulates due to lack of exercise, day sleep; intake of sweet, unctous and slimy food; curd; milk; meat of aquatic & marshy animals.	Simultaneous acumulation of all the *Doṣās* due to intake of unwholesome, uncooked, mutually contradictory & heavy food during weak power of digestion; when unwholesome ingredients like menstrual blood, hair, stool, urine, bone, nails etc. are administered by a woman and slow poisoning (*duṣiviṣa*).
Clinical features	Swelling in the belly, hands, feet and scrotum, tearing of abdomen; increase & decrease of the swelling in the abdomen without any apparent cause; colic pain in the belly & sides	Burning sensation, fever, thirst, fainting, diarrhoea, giddiness, pungent taste in the mouth,	Heaviness, anorexia, indigestion & malaise, numbness, appearance of swelling in hands, legs, scrotum and thighs, nausea, excessive sleep, cough, dyspnoea,	Appearance of signs & symptoms of all the three *Doṣās*
	Appearance of blackish or reddish tinge in nails, eyes, face, skin, urine & stool	Appearance of green & yellow tinge in nails, eyes, face, skin, urine & stool	Appearance of whiteness of nails, eyes, face, skin, urine & stool	Morbidity of nails etc. with all the types of colours;
	Appearance of thin & black net work of veins over the abdomen	Appearance of blue, yellow, deep yellow, green or coppery streaks & veins on abdomen	Appearance of white streaks & veins all over the abdomen	Appearance of network of veins over the abdomen having various colours and streaks in the abdomen.
	On percussion, sounds like the beat of an inflated leather bag and movement of *Vāta* upwards, downwards & sidewards alongwith colic pain & sound.	Suffers from burning sensation, pain, fuming, heating, sweating, moistening & is soft on palpation & prone to immediate inflammation	Abdomen becomes heavy, timid, immobile & hard.	This disease becomes excacerbated espescially during the time of cold breeze & cloudy sky, burning sensation, fainting, anaemia, emaciation & thirst. This condition is also called *Dūṣyodara* & it is dreadful.
Contemporary	Abdominal tuberculosis, Chronic colitis	Acute colitis	Abdominal malignancy	Lead poison

MCQs

1. *Dushyodara* is
   - a. *Sannipata-Udara*
   - b. *Chidrodara*
   - c. *Baddhodara*
   - d. *Jalodara*
2. *Nabheruparigopuchaakriti* is seen in
   - a. *Pleehodara*
   - b. *Jalodara*
   - c. *Kshatodara*
   - d. *Baddhodara*
3. Which *srotas* is involved in *udararoga*
   - a. *Udakavaha*
   - b. *Swedavaha*
   - c. *Udaka & sweadavaha*
   - d. *Anna vaha*
4. Arrange the following in the ascending order of prognosis
   - a. Vatodara<Pittodara<Kaphodara< Pleehodara< Sannipatodara<Udakodara
   - b. Vatodara<Pittodara< Pleehodara <Kaphodara<Sannipatodara<Udakodara
   - c. Vatodara<Pittodara<Kaphodara< Sannipatodara <Pleehodara <Udakodara
   - d. Vatodara<Pittodara<Kaphodara< Udakodara <Pleehodara<Sannipatodara
5. *Shlaksnagandatwam* is the *lakshana* of
   - a. *Rajayakshma*
   - b. *Gulma*
   - c. *Arsas*
   - d. *Udara*
6. *Vayu* involved in the *Samprapti* of *Udara*
   - a. *Prana & agni*
   - b. *Apana & Samana*
   - c. *Vyana & Udana*
   - d. *Prana, Agni & Apana*
7. *Adhmathadhrithitsabdhavadbhavati* is the *lakshana* of
   - a. *Vatikaudara*
   - b. *Jalodara*
   - c. *Vatikavridhi*
   - d. Both a & c
8. *Garadushivisha* are considered as the *nidana* of
   - a. *Pleehodara*
   - b. *Sannipatodara*
   - c. *Badhodara*
   - d. *Kshatodara*
9. सोऽष्ठीलेवातिकठिनः प्राक्ततः कूर्मपृष्ठवत् । are the features of
   - a. *Vatikaudara*
   - b. *Kaphodara*
   - c. *Pleehodara*
   - d. *Badhodara*
10. जन्मनै सर्वं प्रायः कृच्छ्रतमं मतम् । fill the blank
    - a. *Kamala*
    - b. *Udara*
    - c. *Pandu Roga*
    - d. *Grahani roga*
11. बलिनस्तदजाताम्ब यत्रसाध्यंनवोत्थितम् prognosis mentioned in context to
    - a. *Kamala*
    - b. *Udara*
    - c. *Pandu Roga*
    - d. *Grahani roga*

Answer

1.	a	2.	d	3.	c
4.	a	5.	d	6.	d
7.	a	8.	b	9.	c
10.	b	11.	b		

CHAPTER
28

Hepatobiliary diseases

Liver cirrhosis

- Cirrhosis is defined histologically as a diffuse hepatic process characterized by fibrosis and conversion of the normal liver architecture into structurally abnormal nodules.
- The progression of liver injury to cirrhosis may occur over several weeks to years.

Signs and symptoms

- Some patients with cirrhosis are completely asymptomatic and have a reasonably normal life expectancy.
- Other individuals have a multitude of the most severe symptoms of end-stage liver disease and a limited chance for survival.
- Common signs and symptoms may stem from decreased hepatic synthetic function (eg, coagulopathy), portal hypertension (eg, variceal bleeding), or decreased detoxification capabilities of the liver (eg, hepatic encephalopathy).

Portal hypertension

- Portal hypertension can have prehepatic, intrahepatic, or posthepatic causes.

Budd-Chiari syndrome, a posthepatic cause, is characterized by the following symptoms:

- Hepatomegaly
- Abdominal pain

Ascites

Ascites is suggested by the following findings on physical examination:

- Abdominal distention
- Bulging flanks
- Shifting dullness
- Elicitation of a "puddle sign" in patients in the knee-elbow position

Hepatic encephalopathy

The symptoms of hepatic encephalopathy may range from mild to severe and may be observed in as many as 70% of patients with cirrhosis. Symptoms are graded on the following scale:

Grade 0 - Subclinical; normal mental status but minimal changes in memory, concentration, intellectual function, coordination

Grade 1 - Mild confusion, euphoria or depression, decreased attention, slowing of ability to perform mental tasks, irritability, disorder of sleep pattern (ie, inverted sleep cycle)

Grade 2 - Drowsiness, lethargy, gross deficits in ability to perform mental tasks, obvious personality changes, inappropriate behavior, intermittent disorientation (usually with regard to time)

Grade 3 - Somnolent, but arousable state; inability to perform mental tasks; disorientation with regard to time and place; marked confusion; amnesia; occasional fits of rage; speech is present but incomprehensible

Grade 4 - Coma, with or without response to painful stimuli

- Findings on physical examination in hepatic encephalopathy include asterixis and fetor hepaticus.

Additional signs and symptoms

- Many patients with cirrhosis experience fatigue, anorexia, weight loss, and muscle wasting.
- Cutaneous manifestations of cirrhosis include jaundice, spider angiomata, skin telangiectasias ("paper money skin"), palmar erythema, white nails, disappearance of lunulae, and finger clubbing, especially in the setting of hepatopulmonary syndrome.

Ancillary investigations

Hepatorenal syndrome

- Hepatorenal syndrome is diagnosed when a creatinine clearance rate of less than 40 mL/

min is present or when a serum creatinine level of greater than 1.5 mg/dL, a urine volume of less than 500 mL/day, and a urine sodium level of less than 10 mEq/L are present.

- Urine osmolality is greater than plasma osmolality.

Portal hypertension

- During angiography, a catheter is placed selectively via either the transjugular or transfemoral route into the hepatic vein to measure portal pressure.

Hepatic encephalopathy

- An elevated arterial or free venous serum ammonia level is the classic laboratory abnormality reported in patients with hepatic encephalopathy.
- Electroencephalography may be helpful in the initial workup of a patient with cirrhosis and altered mental status, when ruling out seizure activity may be necessary.
- Computed tomography (CT) scanning and MRI studies of the brain may be important in ruling out intracranial lesions when the diagnosis of hepatic encephalopathy is in question.

Ascites

- Paracentesis is essential in determining whether ascites is caused by portal hypertension or by another process.

Alcoholic Liver Disease

Alcohol is one of the most common causes of chronic liver disease. In the UK mortality from alcoholic liver disease is rising, with over 3000 deaths / year; the mean age at presentation is falling.

The risk alcoholic liver disease is variable and not everyone who drinks heavily will develop liver disease. Only 10% of alcoholics have evidence of cirrhosis at postmortem. Alcoholic liver disease does not occur below a threshold of 21 units/week in women and 28 units/week in men. Most individuals with liver disease will have drunk heavily for more than 5 years.

Although the average alcohol consumption of an individual with cirrhosis is 160 g/day for an average of 8 years, there is no clear linear relationship between done and liver damage.

Some of the risk factors for alcoholic liver disease are :

- Drinking patterns - The type of beverage does not affect risk, but liver damage is more likely to occur in continuous rather than being drinkers.
- Gender - The incidence of alcoholic liver disease is increasing in women, who often conceal alcohol misuse and have higher blood ethanol levels than men after drinking because their body mass is lower.
- Genetic - Alcoholism is more common in monozygotic than dizygotic twins. However, polymorphisms in the genes involved in alcohol metabolism, such as aldehyde dehydrogenase (ALD), have yet to be linked to alcoholic liver disease.
- Nutrition - Animals given a choline-deficient diet are more likely to develop alcoholic liver disease.

Pathophysiology

Alcohol is metabolised almost exclusively by the liver via one of two main pathways.

- Eighty percent of alcohol is metabolised to acetaldehyde by the mitochondrial enzyme, alcohol dehydrogenase (ADH). Acetaldehyde forms adducts with cellular proteins in hepatocytes which activate the immune system, leading to cell injury. Acetaldehyde is then metabolised to acetyl-CoA and acetate by ALD. This generates NADH from NAD (nicotinamide adenine dinucleotide), which changes the redox potential of the cell.
- The remaining 20% is metabolised by the mixed function oxidase enzymes of the smooth endoplasmic reticulum. Cytochrome CYP2E1 is an enzyme which oxidise ethanol to acetate. It is induced by alcohol, and during metabolism of ethanol it releases oxygen free radicals, leading to lipid

peroxidation which can induce mitochondrial damage. The CYP2E1 enzyme also metabolises acetaminophen and hence chronic alcoholics are more susceptible to hepatotoxicity from low doses of paracetamol.

It is thought that pro-inflammatory cytokines may also be involved in inducing hepatic damage in alcoholic hepatitis, since endotoxin is released into the blood because of increased gut permetability, leading to release of TNF-α, IL-1, IL-2 and IL-8 from immune cells. All of these cytokines have been implicated in the pathogenesis of liver fibrosis.

In about 80% of patients with severe alcoholic hepatitis, cirrhosis will coexist at presentation. Iron deposition is common and does not necessarily indicate haemochromatosis. The histological features of alcoholic liver disease, which are identical to those of non-alcoholic steatohepatitis.

Clinical features

Alcoholic liver disease has a wide clinical spectrum ranging from mild abnormalities of LFTs on biochemical testing to advanced cirrhosis. The liver is often enlarged in alcoholic liver disease even in the presence of cirrhosis. Peripheral stigmata of chronic liver disease, such as palmar erythema, are more common in alcoholic cirrhosis than in cirrhosis of other aetiologies. Alcohol misuse may also cause damage of other organs and this should be specifically. Three types of alcoholic liver disease are recognised, but in reality these overlap considerably, as do the pathological changes seen in the liver.

Pathological features of alcoholic liver disease—

- Alcoholic hepatitis
- Macrovesicular steatosis
  Lipogranuloma
- Fibrosis and cirrhosis
  Neutrophil infiltration
- Central hyaline sclerosis
  Mallory's hyaline
  Pericellular fibrosis

23.48 Clincial syndromes of alcoholic liver disease—

Fatty liver

- Asymptomatic abnormal
- Normal/large liver
  liver biochemistry

Alcoholic hepatitis

- Jaundice
- Features of portal
- Malnutrition hypertension (e.g. ascites,
- Hepatomegaly
  encephalopathy)

Cirrhosis

- Stigmata of chronic liver
- Large, normal or small liver disease
- Hepatocellular carcinoma
- Ascites/varices/ encephalopathy

Alocoholic fatty liver disease (AFLD) - This usually presents with elevated transaminases in the absence of hepatomegaly. It has a good prognosis and steatosis usually disappears after 3 months of abstinence.

Alocoholic hepatitis - This presents with jaundice and hepatomegaly; complications of portal hypertension may also be present. It has a significantly worse prognosis than AFLD. About one-third of patients die in the acute episode, particularly those with hepatic encephalopathy or a prolonged prothrombin time. Cirrhosis often coexists; if not present, it is the likely outcome if drinking continues. Patients with acute alcoholic hepatitis often deteriorate during the first 1-3 week in hospital. Even if they abstain, it may take up to 6 months for jaundice to resolve. In patients presenting with jaundice who subsequently abstain, the 3-and 5-year survival is 70%. In contrast, those who continue to drink have 3-and 5-year survival rates of 60% and 34% respectively.

Alcoholic cirrhosis—Alcohol-induced cirrhosis often presents with a serious complication such as variceal hemorrhage or ascites, and only half of such patients will survive 5 years from presentation. However, most who survive the initial illness and who become abstinent will survive beyond 5 years.

Ancillary Investigations

Investigations aim to establish alcohol misuse, exclude alternative or coexistent causes of liver disease, such as hepatitis C or haemochromatosis, and assess the severty of liver disease. The clinical history from patient, relatives and friends is most improtant in establishing alcohol misuse, duration and soverity. Biological markers, particularly macrocytosis in the absence of anaemia, may suggest and support a history of alcohol misuse. A raised CGT is not specific for alcohol misuse and will be elevated in the presence of hepatic steatosis and fibrosis. The lvel may not therefore retun to normal with abstinence if chronic liver disease is present. Unexplained ribfractures, particularly bilateral, on a chest X-ray are also suggestive of alcohol misuse. The presence of jaundice suggests alcoholic hepatitis. Determining the extent of liver damage often requires a liver biopsy.

Prothrombin time and bilirubin are used to give a 'discriminant function' (DF), also known as the Maddrey score, which enables the clinician to assess prognosis in alcoholic hepatitis.

Nonalcoholic Fatty Liver Disease (NAFLD)

NAFLD includes simple fatty infiltration (a benign condition called fatty liver) and nonalcoholic steatohepatitis (NASH), a less common but more important variant. NASH (sometimes called steatonecrosis) is diagnosed most often in patients between 40 and 60 years but can occur in all age groups. Many affected patients have obesity, type 2 diabetes mellitus (or glucose intolerance), dyslipidemia, and/or metabolic syndrome.

Pathophysiology of NAFLD

- Fatty liver develops for many reasons, involves many different biochemical mechanisms, and causes different types of liver damage.
- Pathophysiology involves fat accumulation (steatosis), inflammation, and, variably, fibrosis. Steatosis results from hepatic triglyceride accumulation.
- Possible mechanisms for steatosis include reduced synthesis of very low density lipoprotein (VLDL) and increased hepatic triglyceride synthesis (possibly due to decreased oxidation of fatty acids or increased free fatty acids being delivered to the liver).
- Inflammation may result from lipid peroxidative damage to cell membranes. These changes can stimulate hepatic stellate cells, resulting in fibrosis. If advanced, NASH can cause cirrhosis and portal hypertension.

Symptoms and Signs of NAFLD

- Most patients are asymptomatic. However, some have fatigue, malaise, or right upper quadrant abdominal discomfort.
- Hepatomegaly develops in about 75% of patients.
- Splenomegaly may develop if advanced hepatic fibrosis is present and is usually the first indication that portal hypertension has developed.
- Patients with cirrhosis due to NASH can be asymptomatic and may lack the usual signs of chronic liver disease.

Ancillary investigations

- History (presence of risk factors, absence of excessive alcohol intake)
- Serologic tests that rule out hepatitis B and C
- Ultrasound evidence of steatosis or MR elastography with fat fraction

Liver biopsy

- The diagnosis of NASH should be suspected in patients with metabolic syndrome (typically obesity, type 2 diabetes mellitus or a high fasting plasma glucose level, hypertension, and dyslipidemia) and in patients with unexplained laboratory abnormalities suggesting liver disease.
- Differentiating simple steatosis from NASH can be difficult and elevated liver enzymes are not a sensitive predictor for identifying NASH.
- The presence of metabolic syndrome as well as elevated ferritin increases the likelihood that

a patient has NASH rather than simple steatosis.

- When liver enzymes are elevated the most common laboratory abnormalities are elevations in aminotransferase levels.
- Unlike in alcohol-related liver disease, the ratio of aspartate aminotransferase (AST)/alanine aminotransferase (ALT) in NASH is usually < 1.
- Alkaline phosphatase and gammaglutamyl transpeptidase (GGT) occasionally increase.
- Hyperbilirubinemia, prolongation of prothrombin time (PT), and hypoalbuminemia are uncommon.
- For diagnosis, strong evidence (such as a history corroborated by friends and relatives) that alcohol intake is not excessive (eg, is < 20 g/day) is needed,
- Serologic tests should show absence of hepatitis B and C (ie, hepatitis B surface antigen and hepatitis C virus antibody should be negative).
- Liver biopsy reveals damage similar to that seen in alcoholic hepatitis, usually including large fat droplets (macrovesicular fatty infiltration) as well as pericellular or "chicken wire" fibrosis.
- Indications for biopsy include unexplained signs of portal hypertension (eg, splenomegaly, cytopenia) and unexplained elevations in aminotransferase levels that persist for > 6 months in a patient with diabetes, obesity, or dyslipidemia.
- Liver imaging tests, including ultrasonography, CT, and particularly MRI, may identify hepatic steatosis.
- Noninvasive measures of fibrosis such as transient elastography (a test that uses both ultrasound and low-frequency elastic waves), ultrasound elastography, or MR elastography can assess severity of steatosis as well as estimate fibrosis, thereby obviating the need for liver biopsy in many cases
- Transient elastography and ultrasound elastography can be limited by body habitus (too big/fat for ultrasound waves to penetrate adequately), whereas MR elastography is not. However, these tests cannot identify the inflammation typical of NASH and cannot differentiate NASH from other causes of hepatic steatosis.

Hepatitis

Hepatitis is a common cause of jaundice and must be considered in anyone presenting with hepatitic liver blood tests (high transaminases).

All these viruses cause illnesses with similar clinical and pathological features and which are frequently anicteric or even a symptomatic. They differ in their tendency to cause acute and chronic infections.

Causes of viral hepatitis

Common—

- Hepatitis A
- Hepatitis B ± hepatitis D
- Hepatitis C
- Hepatitis E

Less common—

- Cytomegalovirus
- Epstein-Barr virus

Rare—

- Herpes simplex
- Yellow fever

Features of the main hepatitis viruses

	Hepatitis A	Hepatitis B	Hepatitis C	Hepatitis D	Hepatitis E
Virus					
Group	Enterovirus	Hepadna virus	Flavivirus	Incomplete virus	Calicivirus
Nucleic acid	RNA	DNA	RNA	RNA	RNA
Size (diameter)	27 non	42 nm	30-38 nm	35 nm	27 nm

Incubation (weeks)	2-4	4-20	2-26	6-9	3-8
Spread faeces	Yes	No	No	No	Yes
Blood	Uncommon	Yes	Yes	Yes	No
Saliva	Yes	Yes	Yes	Unknown	Unknown
Sexual	Uncommon	Yes	Uncommon	Yes	Unknown
Vertical	No	Yes	Uncommon	Yes	No
Chronic infection	No	Yes	Yes	Yes	No
Prevention					
Active	Vaccine	Vaccine	No	Prevented by hepatitis	No
Passive	Immune serum globulin	Hyperimmune serum globulin	No	B vaccination	No

Note : All body fluids are potentially infectious, although some (i.g. urine) are less infectious than others.

Clinical features of acute infection

Non-specific prodromal illness characterised by headache, myalgia, arthralgia, nausea and anorexia usually precedes the development of jaundice by a few days to 2 weeks. Vomiting and diarrhoea may follow, and abdominal discomfort is common. Dark urine and pale stools may precede jaundice. There are usually few physical signs. The liver is often tender but only minimally enlarged. Occasionally, mild splenomegaly and cervical lymphadenopathy are seen. These are more frequent in children or those with Epstein-Barr virus infection.

Jaundice may be mild and the diagnosis may be suspected only after finding abnormal liver blood tests. Symptoms rarely last longer than 3-6 weeks.

Complications of acute viral hepatitis

- Acute liver failure
- Chronic liver disease and cirrhosis (hepatitis B and C)
- Cholestatic hepatitis (hepatitis A)
- Relapsing hepatitis
- Aplastic anaemia

Investigations

A hepatitic pattern of Liver function tests develops, With serum transaminases typically between 200 and 2000 U/L. The plasma bilirubin reflects the degree of liver damage. The ALP rarely exceeds twice the upper limit of normal. Prolongation of the prothrombin time indicates the severity of the hepatitis but, except in rare cases of acute liver failure, rarely exceeds 25 seconds. The white cell count is usually normal with a relative lymphocytosis. Serological tests confirm the aetiology of the infection.

Hepatitis A

The hepatitis A virus (HAV) belongs to the picornavirus group of enteroviruses. HAV is highly infectious and is spread by the faecal-oral route. Infected individuals, who may be a symptomatic, excrete the virus in faeces for about 2-3 weeks before the onset of symptoms and then for a further 2 weeks or so. Infection is common in children but often a symptomatic, and so up to 30% of adults will have serological evidence of post infection but give no history of jaundice. Infection is also more common in areas of overcrowding and poor sanitation. In occasional outbreaks water and shellfish have been the vehicles of transmission.

In contrast to hepatitis B, a chronic carrier state does not occur.

Investigations

Only one HAV antigen has been found; individuals infected with HAV make an antibody to this antigen (anti-HAV). Anti-HAV is important in diagnosis, as HAV is only present in the blood transiently during the incubation period. Excretion in the stools occurs for only 7-14 days after the onset of the clinical illness and the virus cannot be grown readily. Anti-HAV of the IgM type indicating a primary immune response, is already present in the blood at the onset of the clinical illness and is diagnostic of an acute HAV infection. Titres of this anti-body fall to low levels within about 3 months of recovery. Anti-HAV infection is common and this antibody persists for year after infection, but it can be used as a marker of previous HAV infection. Its presence indicates immunity to HAV.

Hepatitis B

The hepatitis B virus consists of a core containing DNA and a DNA polymerase enzyme needed for virus replication. The core of the virus is sorrounded by surface protein. The virus, also called a Dane particle, and an excess of its surface protein (known as hepatitis B surface antigen) circulate in the blood. Humans are the only source of infection.

Hepatitis B infection affects 300 million people and is one of the most common cuase of chronic liver disease and hepatocellular carcinoma world-wide.

Hepatitis B may cause an acute viral hepatitis; however, the acute infection is often asymptomatic particularly when acquired at birth. Many individuals with chronic hepatitis B are also asymptomatic. Chronic hepatitis, associated with elevated serum transaminases, may occur and can lead to cirrhosis, usually after decades of infection.

The risk of progression to chronic liver disease depends on the source of infection. Vertical transmission from mother to child in the perinatal period is the most common cause of infection world-wide and carries the highest risk.

In this setting, adaptive immune responses to HBV may be absent initially, with apparent immunological tolerance. Several mechanisms contribute towards this tolerance.

- Firstly, the introduction of antigen in the neonatal period is tolerogenic.
- Secondary, the presentation of such antigen within the liver, as described above, promotes tolerance; this is particularly evident in the absence of a significant innate or inflammatory response.
- Finally, very high loads of antigen may lead to so called exhaustion of cellular immune responses.

However, the state of tolerance is not permanent and may be reversed as a result of therapy, or through spontaneous changes in innate responses such as interferon-alpha and NK cells, accomparied by host mediated immunopathology.

Investigation

Serology—HBV contains several antigens to which infected persons can make immune responses; these antigens and their antibodies are important in identifying HBV infection .

- *The hepatitis B surface antigen* (HBsAg) is an indicator of active infection, and a negative test for HBsAg makes HBV infection very unlikely. In acute liver failure from hepatitis B and liver damage is mediated by viral clearance and so HBsAg is negative, with evidence of recent infection shown by the presence of hepatitis B core IgM. HBsAg appears in the blood late in the incubation period but before the prodromal phase of acute type B hepatitis, it may be present for a few days only disappearing even before jaundice has developed, but usually last for 3-4 weeks and can persist for up to 5 months indicates chronic infection. Antibody to HBsAg (anti-HBs) usually appears after about 3-6 months and persists for many years or perhaps permanently. Anti-HBs implies either a previous infection, in which case anti-HBc is usually also present, or previous vaccination, in which case anti HBc is not present.

- *The hepatitis B core antigen* (HBcAg) is not found in the blood, but antibody to it (anti-HBc) appears early in the illness and rapidly reaches a high titre, which subsides gradually but then persists. Anti-HBc is initially of IgM type with IgG antibody appearing later. Anti-HBc (IgM) can sometimes reveal an acute HBV infection when the HBsAg has disappeared and before anti-HBs has developed.
- *The hepatitis B e antigen* (HBeAg) is an indicator of viral replication. In acute hepatitis B it may appear only transiently at the outset of the illness; its apearance is followed by the production of antibody (anti-HBe). The HBeAg reflects active replication of the virus in the liver.

Chronic HBV infection is marked by the presence of HBsAg or anti-HBc (IgG) in the blood. Usually, HBeAg or anti-HBe is also present; HBeAg indicates continued active replication of the virus in the liver. Although the presence of anti-HBe usually implies low viral replication, the exception is HBeAb-positive replicating chronic hepatitis B (also called 'pre-core mutant infection), in which high levels of serum HBV-DNA are seen, despite negative HBeAg.

Viral load—HBV-DNA can be measured by polymerase chain reaction (PCR) in the blood. Viral loads are usually in excess of 10^5 copies/mL in the presence of active viral replication, as indicated by the presence of e antigen. In contrast, in those with low viral replication, HBsAg and anti-HBe-positive, viral loads are less than 10^5 copies mL. The exception is in patients who have a mutation in the pre-core protein, which means they cannot secrete antigen into serum. Such individuals will be anti-HBe-positive but have a high viral load and often evidence of chronic hepatitis. These mutations are common in the Far East and those patients affected are classified as having e antigen-negative chronic hepatitis. They respond differently to antiviral drugs from those with classical e antigen-positive chronic hepatitis.

Measurement of viral load is important in monitoring antiviral therapy and identifying patients with pre-core mutants. Specific HBV genotypes can also be identified using PCR. Genotypes B and C appear to have more aggressive disease that responds less well to interferon.

Hepatitis D (Delta virus)

The hepatitis D virus (HDV) is an RNA-defective virus which has no independent existence; it requires HBV for replication and has the same sources and modes of spread. It can infect individuals simultaneously with HBV, or can superinfect those who are already chronic carriers of HBV Simultaneous infections give rise to acute hepatitis, which is often severe but is limited by recovery from the HBV infection. Infections in individuals who are chronic carriers of HBV can cause acute hepatitis with spontaneous recovery, and occasionally simultaneous cessation of the chronic HBV infection occurs. Chronic infection with HBV and HDV can also occur, and this frequently causes rapidly progressive chronic hepatitis and eventually cirrhosis.

HDV has a world-wide distribution. It is endemic in arts of the Mediterranean basin, Africa and South America, where transmission is mainly by close personal contact and occasionally by vertical transmission from mothers who also carry HBV. In no-endemic areas, transmission is mainly a consequence of parenteral drug misuse.

Investigations

HDV contains a single antigen to which infected individuals make an antibody (anti-HDV). Delta antigen appears in the blood only transiently, and in practice diagnosis depends on detecting anti-HDV. Simultaneous infection with HBV and HDV followed by full recovery is associated with the appearance of low titers of anti-HDV of IgM type within a few days of the onset of the illness. This antibody generally disappears within 2 months but persists in a few patients. Superinfection of patients with chronic HBV infection leads to the production of high titers of anti-HDV, initially IgM and later IgG. Such patients may then develop chronic infection with both viruses, in which case anti-HDV titres plateau at high levels.

Hepatitis C

This is caused RNA flavivirus. Acute symptomatic infection with hepatitis C is rare. Most individuals are unaware of when they became infected and only identified when they develop chronic liver disease.

Eighty per cent of individuals exposed to the virus become chronically infected and late spontaneous viral clearance is rare.

Hepatitis C is the cause of what used to be known as 'non-A, non-B hepatitis', a syndrome of acute hepatitis often with jaundice seen after a transfusion of blood or blood products. Following the identification of the virus In 199Q, blood donors are now screened for infection in many parts of the world. New cases of post- transfusion hepatitis C no longer occur in the UK.

Hepatitis C infection is usually now identified in asymptomatic individuals screened because they have risk factors for infection such as previous injection drug use or they have incidentally been found to have abnormal liver blood tests. Although most individuals remain asymptomatic until progression to cirrhosis occurs, fatigue can complicate chronic infection and appears to be unrelated to the degree of liver damage.

Investigations

Serology and virology—The HCV protein contains several antigens that give rise to antibodies in an infected person, and these are used in diagnosis. It may take 6-12 weeks for antibodies to appear in the blood following acute infection such as a needlestick injury. In these cases hepatitis C RNA can be identified in the blood as early as 2-4 weeks after infection. Active infection is confirmed by the presence of serum hepatitis C RNA in anyone who is antibody-positive. Anti-HCV antibodies persist in serum even after viral clearance, whether spontaneous or post-treatment.

Molecular analysis—There are six common viral genotypes whose distribution varies worldwide. Genotype has no effect on progression of liver disease but does affect response to treatment. Genoype 1 is most common in northern Europe and is less easy to eradicate with current treatment.

Risk factors for the acqusition of chronic hepatitis C infection—

- Intravenous drug misuse (95% of new cases in the UK)
- Unscreened blood products
- Vertical transmission (3% risk)
- Needlestick injury (3% risk)
- Iatrogenic parenteral transmission, (i.e. contaminated vaccination needles)
- Sharing toothbrushes/razors

Liver function tests—LFTs may be normal or show fluctuating serum transaminases between 50 and 200 U/L. Jaundice is rare and only usually appears in end-stage cirrhosis.

Liver histology—Serum transaminase levels in hepatitis C are a poor predictor of the degree of liver fibrosis, and so a liver biopsy is often required to stage the degree of liver damage. Non-invasive methods of assessing liver fibrosis in hepatitis C infection remain an area of active research. The degree of inflammation and fibrosis can be scored histologically. The most common scoring system use din hepatitis C is the Metavir system, which scores fibrosis from 1 to 4, the latter equating to cirrhosis.

Hepatitis E

Hepatitis E is caused by an RNA virus which is endemic in India and the Middle East. An increase in prevalence has recently been noted in northern Europe and infection is no longer seen only in travellers from an endemic area.

The clinical presentation and management of hepatitis E are similar to that of hepatitis A. Disease is spread via the faecal-oral route; in most cases, it presents as a self-limiting acute hepatitis and does not cause chronic liver disease.

Hepatitis E differs from hepatitis A in that infection during pregnancy is associated with the development of acute liver failure, which has a high mortality. In acute infection, IgM antibodies to HEV are positive.

Other forms of viral hepatitis

Non-A, non-B, non-C (NANBNC) or non-A-E hepatitis is the term used to describe hepatitis thought to be due to a virus that is not HAV, HBV, HCV or HEV. Other viruses which affect the liver probably do exist, but the hepatitis viruses described above now account for the majority of hepatitis virus infections. Cytomegalovirus and Epstein-Barr virus infection causes abnormal LFTs in most patients, and occasionally icteric hepatitis occurs. Herpes simplex is a rare cause of hepatitis in adults, and most of these patients are immunocompromised. Abnormal LFTs are also common in chickenpox, measles, rubella and acute HIV infection.

Causes of abnormal liver blood tests in HIV infection—

Hepatitic blood tests—

- Chronic hepatitis C
- Antiretroviral drugs
- Chronic hepatitis B
- Cytomegalovirus

Cholestatic blood tests—

- Tuberculosis
- Sclerosing cholangitis due to Cryptosporidia
- Atypical mycobacterium

Jaundice

1. Jaundice is usually detectable clinically when the plasma bilirubin exceeds 50 mol/L (-3 mg/dL).
2. The causes of jaundice overlap with the causes of abnormal LFTs.
3. In a patient with jaundice it is useful to consider whether the cause might be pre-hepatic, hepatic or post-hepatic.

Common causes of elevated serum transaminases

Minor elevation (< 100 U/L)

- Chronic hepatitis C
- Haemochromatosis
- Chronic hepatitis B
- Fatty liver disease

Moderate elevation (100-300 U/L)

As above plus:

- Alcoholic hepatitis
- Autoimmune hepatitis
- Non-alcoholic steatohepatitis
- Wilson's disease

Major elevation (> 300 U/L)

- Drugs (e.g. paracetamol)
- Ischaemic liver
- Acute viral hepatitis
- Toxins (e.g. *Amanita phalloides* poisoning)
- Autoimmune liver disease
- Flare of chronic hepatitis B

Pre-Hepatic Jaundice

1. This is caused either by haemolysis or by congenital hyperbilirubinaemia, and is characterised by an isolated raised bilirubin level.
2. In haemolysis, destruction of red blood cells or their precursors in the marrow causes increased bilirubin production.
3. Jaundice due to haemolysis is usually mild because a healthy liver can excrete a bilirubin load six times greater than normal before unconjugated bilirubin accumulates in the plasma.
4. The newborn, who have a reduced capacity to metabolise bilirubin .Hence this rule does not apply.
5. The most common form of non-haemolytic hyperbilirubinaemia is Gilbert's syndrome, an inherited disorder of bilirubin metabolism
6. Other inherited disorders of bilirubin metabolism also exist but they are very rare.

Congenital non-haemolytic hyperbilirubinaemia

Syndrome	Inheritance	Abnormality	Clinical features/treatment
Unconjugated hyperbilirubinaemia			
Gilbert's	Autosomal dominant	Glucuronyl transferase Bilirubin uptake	Mild jaundice, especially with fasting No treatment necessary
Crigler-Najjar			
Type I	Autosomal recessive	Absent glucuronyl transferase	Rapid death in neonate (kernicterus)
Type II	Autosomal dominant	Absent glucuronyl Glucuronyl transferase	Presents in neonate Phenobarbital, ultraviolet light or liver transplant as treatment
Conjugated hyperbilirubinaemia			
Dubin-Johnson	Autosomal recessive	Canalicular excretion of organic anions, including bilirubin	Mild No treatment necessary
Rotor's	Autosomal recessive	Bilirubin uptake Intrahepatic binding	Mild No treatment necessary

Hepatocellular Jaundice

1. Hepatocellular jaundice results from an inability of the liver to transport bilirubin across the hepatocyte into the bile, occurring as a consequence of parenchymal liver disease.
2. Bilirubin transport across the hepatocytes may be impaired at any point between uptake of unconjugated bilirubin into the cells and transport of conjugated bilirubin into the canaliculi.
3. The swelling of cells and oedema resulting from the disease itself may cause obstruction of the biliary canaliculi.
4. In hepatocellular jaundice the concentrations of both unconjugated and conjugated bilirubin in the blood increase.
5. Hepatocellular jaundice can be due to acute or chronic liver injury.
6. AST- ALT; GGT, ALP : Characteristically, jaundice due to parenchyma liver disease is associated with increases in transaminases (AST, ALT), but increases in other LFTs, including cholestatic enzymes (GGT, ALP) may occur, and suggest specific aetiologies.
7. AST > 1000 U/L : Acute jaundice in the presence of AST > 1000 U/L is highly suggestive of an infectious cause (e.g. hepatitis A, B), drugs (e.g. paracetamol) or hepatic ischaemia.
8. Imaging : Imaging is essential, in particular to identify features suggestive of cirrhosis (e.g. irregular liver outline, splenomegaly), to define the patency of hepatic arteries and veins, and of the portal vein, and to obtain evidence of portal hypertension.
9. Liver biopsy : Liver biopsy has an important role in defining the aetiology of hepatocellular jaundice and the extent of established liver injury.

Obstructive (Cholestatic) Jaundice

Cholestatic jaundice may be caused by:

- Failure of hepatocytes to initiate bile flow
- Obstruction of bile flow in the bile ducts or portal tracts
- Obstruction of bile flow in the extrahepatic bile ducts between the porta hepatis and the papilla of Vater.
- In the absence of treatment, cholestatic jaundice tends to become progressively more

severe because conjugated bilirubin is unable to enter the bile canaliculi and passes back into the blood, and also because there is a failure of clearance of unconjugated bilirubin arriving at the liver cells.

Causes *of* cholestatic jaundice

Intrahepatic

- Primary biliary cirrhosis
- Pregnancy
- Primary sclerosing cholangitis
- Inherited cholestatic liver disease, e.g. benign recurrent intrahepatic cholestasis
- Alcohol
- Drugs
- Cystic fibrosis
- Chronic right heart failure
- Severe bacterial infections
- Hepatic infiltrations (lymphoma, granuloma, amyloid, metastases)

Extrahepatic

- Carcinoma
  Ampullary
  Pancreatic
  Bile duct

- Choledocholithiasis
- Parasitic infection
- Traumatic biliary strictures
- Chronic pancreatitis (cholangiocarcinoma)

Liver metastases

- Cholestasis may result from defects at more than one of these levels.
- Those confined to the extrahepatic bile ducts may be amenable to surgical correction.

Clinical features in cholestatic jaundice

Obstruction of the bile duct drainage due to blockage of the extrahepatic biliary tree :

- Pale stools
- Dark urine
- Pruritus accompanied by skin excoriations.
- Peripheral stigmata of chronic liver disease are absent.

Courvoisier's Law

- If the gallbladder is palpable, the jaundice is unlikely to be caused by biliary obstruction due to gallstones, probably because a chronically inflamed stone-containing gallbladder cannot readily dilate. This is Courvoisier's Law, and suggests that jaundice is due to a malignant biliary obstruction (e.g. pancreatic cancer).

Charcot's triad

- Cholangitis is characterised by 'Charcot's triad' of jaundice, right upper quadrant pain and fever.

ALP and GGT

- Cholestatic jaundice is characterised by a relatively greater elevation of ALP and GGT than the aminotransferases.

Ultrasound

- Ultrasound evaluation is indicated in all cases to determine whether there is evidence of mechanical obstruction and dilatation of the biliary tree .

Clinical features and complications of cholestatic jaundice

Cholestasis	Early features
• Jaundice	• Pale stools
• Dark urine	• Pruritus

Late features :

- Malabsorption (vitamins A, D, E and K)

Weight loss	Steatorrhoea
Osteomalacia	Bleeding tendency

Cholangitis
- Fever
- Pain (if gallstones are present)
- Rigors

Clinical features suggesting an underlying cause of cholestatic jaundice

Clinical feature	Causes
Jaundice Static or increasing	Carcinoma Primary biliary cirrhosis Primary sclerosing cholangitis
Fluctuating	Choledocholithiasis Stricture

	Pancreatitis Choledochal cyst Primary sclerosing cholangitis
Abdominal pain	Choledocholithiasis Pancreatitis Choledochal cyst
Cholangitis	Stone Stricture Choledochal cyst
Abdominal scar	Stone Stricture
Irregular hepatomegaly	Hepatic carcinoma
Palpable gallbladder	Carcinoma below cystic duct (usually pancreas)
Abdominal mass	Carcinoma Pancreatitis (cyst) Choledochal cyst
Occult blood in stools	Ampullary tumour

Ascites

1. Excess accumulation of free fluid in the peritoneal cavity is known as Ascites

Causes of ascites

Common causes

Malignant disease
- Cardiac failure

Hepatic
- Hepatic cirrhosis

Peritoneal

Other causes

Hypoproteinaemia
- Pancreatitis

Nephrotic syndrome
- Lymphatic obstruction

Protein-losing enteropathy

Malnutrition
- Infection Tuberculosis

Hepatic venous occlusion

Budd-Chiari syndrome

Veno-occlusive disease

Rare causes

- Meigs syndrome
- Hypothyroidism

('Meigs' syndrome is the association of a right pleural effusion with or without ascites and a benign ovarian tumour. The ascites resolves on removal of the tumour)

Pathophysiology

Splanchnic vasodilatation is thought to be the main factor leading to ascites in cirrhosis.

↓

This is mediated by vasodilators (mainly nitric oxide) that are released when portal hypertension causes shunting of blood into the systemic circulation.

↓

Systemic arterial pressure falls due to pronounced splanchnic vasodilatation as cirrhosis advances.

↓

This leads to activation of the renin-angiotensin system with secondary aldosteronism, increased sympathetic nervous activity, increased atrial natriuretic hormone secretion and altered activity of the kallikrein-kinin system

↓

These systems tend to normalise arterial pressure but produce salt and water retention.

↓

The combination of splanchnic arterial vasodilatation and portal hypertension alters intestinal capillary permeability,

↓

Promoting accumulation of fluid within the peritoneum

↓

Ascites

Clinical features

1. Small amounts of ascites are asymptomatic
2. Larger accumulations of fluid (> 1 L) produces clinical manifestations
   - Abdominal distension
   - fullness in the flanks

   Shifting dullness
   - Shifting dullness on percussion

   Fluid thrill
   - When the ascites is marked, a fluid thrill is present
3. In obese patients, much larger volumes of

ascites may accumulate before they are detectable clinically.

Other physical features

- Distortion or eversion of the umbilicus
- Herniae
- Abdominal striae
- Divarication of the recti
- Scrotal oedema.
- Dilated superficial abdominal veins may be seen if the ascites is due to portal hypertension.

Investigations

Ultrasonography

1. Ultrasonography is the best means of detecting ascites, particularly in the obese and those with small volumes of fluid

Paracentesis

2. Paracentesis (if necessary under ultrasonic guidance) can also be used to confirm the presence of ascites but is most useful for obtaining ascitic fluid for analysis.

Ascites fluid appearance and analysing

Cause/appearance

- Cirrhosis: clear, straw-coloured or light green
- Malignant disease: bloody
- Infection: cloudy
- Biliary communication: heavy bile staining
- Lymphatic obstruction: milky-white (chylous)

Useful investigations

- Total albumin (plus serum albumin) and protein
- Amylase
- Neutrophil count
- Cytology
- Microscopy and culture

Radiography

- Pleural effusions are found in about 10% of patients, usually on the right side (hepatic hydrothorax); most are small and only identified on chest X-ray, but occasionally a massive hydrothorax occurs.
- Pleural effusions, particularly those on the left side, should not be assumed to be due to the ascites.

Serum-Acites Albumin Gradient (SAAG)

- Measurement of the protein concentration and the serum-ascites albumin gradient (SAAG) are used to distinguish a transudate from an exudate.

Transudate Ascites

- Cirrhotic patients typically develop a transudate with a total protein concentration below 25 g/L and relatively few cells.
- However, in up to 30%, of patients, the total protein concentration is more than 30 g/L.
- In these cases it is useful to calculate the SAAG by subtracting the concentration of the ascites fluid albumin from the serum albumin.
- A gradient of more than 11 g/L is 96% predictive that ascites is due to portal hypertension.
- Venous outflow obstruction due to cardiac failure or hepatic venous outflow obstruction can also cause a transudative ascites, as indicated by an albumin gradient above 11 g/L, but unlike in cirrhosis the total protein content is usually- above 25g/L.

Exudate Ascites

- Ascites protein concentration above 25 g/ L or a SAAG of less than 11 g/ L) raises the possibility of infection (especially tuberculosis), malignancy, hepatic venous obstruction, pancreatic ascites or, rarely, hypothyroidism.

Pancreatic ascites

- Ascites amylase activity above 1000 U/L identifies pancreatic ascites

Ascites glucose concentrations

- Low ascites glucose concentrations suggest malignant disease or tuberculosis.

Cytological examination

- Cytological examination may reveal malignant cells (onethird of cirrhotic patients with a bloody tap have a hepatoma).

Full blood count

- Polymorphonuclear leucocyte counts above 250 x 10^6/L strongly suggest infection (spontaneous bacterial peritonitis).

Laparoscopy

- Laparoscopy can be valuable in detecting peritoneal disease.

Abdominal pain

1. Abdominal pain results from GI disease and extraintestinal conditions involving the genitourinary tract, abdominal wall, thorax, or spine.
2. The most common causes of abdominal pain are irritable bowel syndrome and functional dyspepsia

There are four types of abdominal pain

Visceral pain

- Visceral pain generally is midline in location and vague in character.
- Gut organs are insensitive to stimuli such as burning and cutting but are sensitive to distension, contraction, twisting and stretching.
- Pain from unpaired structures is usually but not always felt in the midline.
- Noninflammatory visceral sources include mesenteric ischemia and neoplasia.

Parietal pain

- It is localized and precisely described .i.e. sharp and lateralised pain
- The parietal peritoneum is innervated by somatic nerves, and its involvement by inflammation, infection or neoplasia
- Common inflammatory diseases with pain include peptic ulcer, appendicitis, diverticulitis, inflammatory bowel disease, and infectious enterocolitis.

Referred pain

- Gallbladder pain -is referred to the back or shoulder tip
- Pancreatitis- The pain radiates directly through the abdomen to the back in approximately one half of cases.

Psychogenic

- Cultural, emotional and psychosocial factors influence everyone's experience of pain.
- In some patients, no organic cause can be found despite investigation, and psychogenic causes (depression or somatisation disorder) may be responsible.

The acute abdomen

This accounts for approximately 50% of all urgent admissions to general surgical units. The acute abdomen is a consequence of one or more pathological processes.

Inflammation

- Pain develops gradually, usually over several hours.
- It is initially rather diffuse until the parietal peritoneum is involved, when it becomes localised.
- Movement exacerbates the pain
- Abdominal rigidity and guarding occur

Perforation

- When a viscus perforates, pain starts abruptly
- It is severe and leads to generalised peritonitis (guarding and rebound tenderness with rigidity)

Obstruction

- Pain is colicky, with spasms which cause the patient to writhe around and double up.
- Colicky pain which does not disappear between spasms suggests complicating inflammation.

Investigations

Blood :

- Full blood count and Urea and electrolytes and amylase taken to look for evidence of dehydration, leucocytosis and pancreatitis.

Erect chest X-ray

- An erect chest X-ray may show air under the diaphragm suggestive of perforation
- Plain abdominal film may show evidence of obstruction

Abdominal ultrasound

- An abdominal ultrasound may help if gall stones or renal stones are suspected.
- It is also useful in the detection of free fluid and any possible intra-abdominal abscess.

Contrast studies

- Contrast studies, by either mouth or anus, are useful in the further evaluation of intestinal obstruction
- Essential in the differentiation of pseudo-obstruction from mechanical large bowel obstruction.

CT abdomen

- To seek an evidence of pancreatitis, retroperitoneal collections or masses, including an aortic aneurysm)

Angiography

To detect mesenteric ischaemia

Diagnostic laparotomy

It should be considered when the diagnosis has not been revealed by other investigations.

Note- All patients must be carefully and regularly reassessed (every 2-4 hours) so that any change in condition which might alter both the suspected diagnosis and clinical decision can be observed and acted upon early.

Causes of acute abdominal pain

Inflammation

- Appendicitis
- Pancreatitis
- Diverticulitis
- Pyelonephritis
- Cholecystitis
- Intra-abdominal abscess
- Pelvic inflammatory disease

Perforation/rupture

- Peptic ulcer
- Ovarian cyst
- Diverticular disease
- Aortic aneurysm

Obstruction

- Intestinal obstruction
- Ureteric colic
- Biliary colic

MCQs

1. Patients with hepatitis typically have an AST: ALT ratio of at least 2:1. The AST rarely exceeds 300 U/L indicates which hepatitis
   a. Alcoholic hepatitis
   b. Viral hepatitis
   c. Drug toxicity hepatitis
   d. Autoimmune hepatitis
2. Which one is correct?

a. Liver biopsy remains the criterion standard in the evaluation of patients withliver disease, particularly in patients with chronic liver diseases & Liver biopsy is necessary for diagnosis but is more often useful in assessingthe severity (grade) and stage of liver damage, in predicting prognosis, and inmonitoring response to treatment.

b. Liver biopsy is not criterion standard in the evaluation of patients with liver disease, particularly in patients with chronic liver diseases & Liver biopsy is necessary for diagnosis but is more often useful in assessing the severity (grade) and stage of liver damage, in predicting prognosis, and in monitoring response to treatment.

c. Liver biopsy is not necessary for diagnosis but is more often useful in assessing the severity (grade) and stage of liver damage, in predicting prognosis, and in monitoring response to treatment.

d. Liver biopsy is not useful in assessing the severity (grade) and stage of liver damage, in predicting prognosis, and in monitoring response to treatment.

3. Itching occurs with acute liver disease observed in

a. Appearing early in obstructive jaundice (from biliary obstruction or drug-Induced cholestasis), somewhat later in hepatocellular disease (acute hepatitis). Itching can occur inany liver disease, particularly once cirrhosis is present & Itching also occurs in chronic liver diseases, typically the cholestatic formssuch as primary biliary cirrhosis and sclerosing cholangitis.

b. Appearing early in hepatocellular jaundice,
c. Appearing early in haemolytic jaundice
d. Appearing early in haemolytic & hepatocellular jaundice

4. An enlarged left supraclavicular node (Virchow's node) or periumbilical nodule (SisterMary Joseph's nodule) suggests
a. Cholecystitis
b. Viral hepatitis
c. Alcoholic hepatitis
d. Abdominal malignancy

5. Ascending cholangitis and biliary sepsisdiagnosed using which triad
a. Anderson Triad b. Beck's triad
c. Charcot's triad d. Dieulafoy's triad

6. Grey turner sign is seen in
a. Acute cholecystitis b. Acute appendicitis
c. Acute pancreatitis d. Acute hepatitis

7. Which of the following physical findings may indicate severe necrotizing pancreatitis?
a. Jaundice
b. Erythematous skin nodules
c. Fever
d. Hypotension

8. Puddle sign is used to detect
a. Ascites
b. Acute cholecystitis
c. Acute appendicitis
d. Abdominal malignancy

9. Hepatorenal syndrome is diagnosed
a. Creatinine clearance rate of less than 60mL/min is present, Serum creatinine level of greater than 2.5 mg/dL & Urine sodiumlevel of less than 10 mEq/L
b. Creatinine clearance rate of less than 70mL/min is present, Serum creatinine level of greater than 3.5 mg/dL & Urine sodiumlevel of less than 10 mEq/L
c. Creatinine clearance rate of less than 80mL/min is present, Serum creatinine level of greater than 4.5 mg/dL & Urine sodiumlevel of less than 10 mEq/L
d. Creatinine clearance rate of less than 40mL/min is present, Serum creatinine level of greater than 1.5 mg/dL & Urine sodiumlevel of less than 10 mEq/L

Answer

1.	a	2.	a	3.	a
4.	c	5.	a	6.	c
7.	b	8.	a	9.	d

CHAPTER 29

Kushtha-Maha Kushtha & Kshudra Kushtha

कुष्ठ (Kuṣṭha)

Introduction

त्वचः कुर्वन्ति वैवर्ण्यं दुष्टाः कुष्ठमुशन्ति तत्।
कालेनोपेक्षितं यस्मात् सर्वं कुष्णाति तद्वपुः॥
A.H.Ni. 14/3-4

The disease *Kuṣṭha* is characterised by discolouration, loss of touch sensation, appearance of rashes, excessive or no perspiration etc. Chronicity of the disease develops deformity and makes the entire body ugly.

कुष्ठ हेतु *(Kuṣṭha Hetu)*

विरोधीन्यन्नपानानि द्रवस्निग्धगुरूणि च।
भजतामागतां छर्दिं वेगांश्चान्यान्प्रतिघ्नताम्॥
व्यायाममतिसंतापमतिभुक्त्वोपसेविनाम्।
शोतोष्णलङ्घनाहारान् क्रमं मुक्त्वा निषेविणाम्॥
घर्मश्रमभयार्तानां द्रुतं शीताम्बुसेविनाम्।
अजीर्णाध्याशिनां चैव पञ्चकर्मापचारिणाम्॥
नवान्नदधिमत्स्यातिलवणाम्लनिषेविणाम्।
माषमूलकपिष्टान्नतिलक्षीरगुडाशिनाम्॥
व्यवायं चाप्यजीर्णेऽति निद्रां च भजतां दिवा।
विप्रान् गुरून् धर्षयतां पापं कर्म च कुर्वताम्॥
C.S.Ci. 7/4-8

मिथ्याहाराचारस्य विशेषाद् गुरुविरुद्धासात्म्याजीर्णाहिताशिनः स्नेहपीतस्य वान्तस्य वा व्यायामग्राम्यधर्मसेविनो ग्राम्यानूपौदकमांसानि वा पयसाऽभीक्ष्णमश्नतो यो वा मज्जत्यप्सूष्माभितप्तः सहसा छर्दिर्वा प्रतिहन्ति। *S.S.Ni. 5/3*

Causative factors of the disease are as follows-

- Consumption of foods and drinks, which are mutually contradictory, liquid, unctuous and heavy. Suppression of natural urges especially vomiting.
- Performance of physical exercise and exposure to intense heat just after taking heavy meals.
- Improper implementation of prescribed order in relation to cold, heat, fasting and diet.
- Usage of cold water just after exposure to intensive sun heat, exertion and fear.
- Indulgence of food during indigestion and intake of food before the digestion of previous meal.
- Improper administration of biopurificatiory procedures i:e *Vamana, Virecana, Basti* and *Nasya Karma.*
- Consumption of freshly harvested grains, curd, fish, salt and sour substances in excess.
- Adequate intake of black gram, radish, flour preparations, sesamum, milk and jaggery.
- Performing sexual intercourse during indigestion.
- Sleeping during day time.
- Indulging in mischievous acts like insulting Brahmins, teachers, elders and other respectful persons, along with other sinful and heinous activities.

Along with above *Suśruta* included following etiological factors.

- Indulging in physical exercise and sexual intercourse after intake of *Sneha* (unctuous substances) or *Vamana Karma* (emesis).
- Regular consumption of meats of domestic, marshy and aquatic animals with milk.
- Immersion in water immediately after having been exposed to heat.
- Sudden suppression of bouts of vomiting

स्त्रीपुंसयोः कुष्ठदोषाद् दुष्टशोणितशुक्रयोः।
यदपत्यं तजोर्जातं ज्ञेयं तदपि कुष्ठितम्॥
S.S.Ni. 5/28

तत्रादिबलप्रवृत्ता ये शुक्रशोणितदोषान्वयाः कुष्ठार्शःप्रभृतयः।
S.S.Su. 24/5

तत्रादिबलप्रवृत्तं पौण्डरीकं काकणं चासाध्यम्।
S.S.Ni. 5/18

Kuṣṭha manifest due to morbidity of *Śukra* and *Śoṇita. Puṇḍarika* and *Kākaṇaka* are caused by *Ādibala pravṛtta* etiology are incurable.

प्रसङ्गाद् गात्रसंस्पर्शान्निश्वासात्सहभोजनात् ।
सहशय्याऽऽसनाच्चापि वस्त्रमाल्यानुलेपनात् ॥
कुष्ठं ज्वरश्च शोषश्च नेत्राभिष्यन्द एव च ।
औपसर्गिकरोगाश्च सङ्क्रामन्ति नरान्नरम् ॥
S.S.Ni. 5/33-34

Kuṣṭha manifest due to following causative factors-

If person adopts following behaviour with *Kuṣṭa* patient then it leads to manifestation of *Kuṣṭha* in that individual.

- Constant close intimacy with the patient
- Touching the patient oftenly
- Inhaling expired air of the patient
- Eating together
- Sleeping and sitting together
- Wearing dress, garlands and unguents used by patient who is suffering from contagious diseases likes *Kuṣṭha, Śoṣa* etc.

In *Aṣṭaṅga Sangraha Kuṣṭha* disease included under *Sahaja Vyādhi A.S.Su. 22/01*

म्रियते यदि कुष्ठेन पुनर्जातेऽपि गच्छति ।
नातः कष्टतरो रोगो यथा कुष्ठं प्रकीर्त्तितम् ॥
S.S.Ni. 5/31

If *Kuṣṭha rogi* dies and if he is born again, it appears in next birth also. That's why it is said to be troublesome disease.

रक्तज क्रिमि

कुष्ठजाः सपरीसर्पा ज्ञेयाः शोणितसम्भवाः ॥
ते सरक्ताश्च कृष्णाश्च स्निग्धाश्च पृथवस्तथा ।
रक्ताधिष्ठानजान् प्रायोविकारान् जनयन्ति ते ॥
S.S.Utt. 54/15-16

Kuṣṭhaja is a subtype of *Raktaja Krimi* and it manifests *Kuṣṭha* disease.

शोणितजानां तु खलु कुष्ठैः समानं ।
C.S.Vi. 7/11

Śoṇitaja Krimi Vikāra manifest due to consumption of *Kuṣṭha* producing causative agents. Treatment similar to *Kuṣṭha roga.*

ब्रह्मस्त्रीसज्जनवधपरस्वहरणादिभिः ।
कर्मभिः पापरोगस्य प्राहुः कुष्ठस्य संभवम् ॥
S.S.Ni. 5/30

Kuṣṭha may manifest due to killing of *Brāhmaṇa,* women, pious persons, robbing the wealth of others and indulging in sinful acts.

कुष्ठ सम्प्राप्ति (Samprāpti of Kuṣṭha)

त्रयो दोषाः युगपत् प्रकोपमापद्यन्ते; त्वगादयश्चत्वारः शैथिल्यमापद्यन्ते; तेषु शिथिलेषु दोषाः प्रकुपित्ताः स्थानमधिगम्य संतिष्ठमानास्तानेव त्वगादीन् दूषयन्तः कुष्ठान्यभिनिर्वर्तयन्ति॥ *C.S.Ni. 5/6*

Due to consumption of *Nidāna tridoṣās* become agitated and brings looseness in *Dhātus [Tvak (Rasa), Rakta, Māmsa* and *Ambu]*. Thus the vitiated *Doṣās* getting accomodation in those tissues, staying there in affect *Tvagādi Dhātus* morbidity leading to manifestation of *Kuṣṭha.*

त्रयो दोषा वातपित्तश्लेष्माणः प्रकोपणविकृताः, दूष्याश्च शरीर- धातवस्त्वङ्मांसशोणितलसीकाश्चतुर्धा दोषोपघात-विकृता इति। एतत् सप्तानां सप्तधातुकमेवङ्गतमाजननं कुष्ठानाम्, अतः प्रभवाण्यभिनिर्वर्तमानानि केवलं शरीरमुपतपन्ति॥ *C.S.Ni. 5/3*

वातादयोस्त्रयो दुष्टास्त्वग्रकतं मांसमम्बु च ।
दूषयन्ति स कुष्ठानां सत्पको द्रव्यसंग्रहः ।
अतः कुष्ठानि जायन्ते सप्त चैकादशैव च ।
न चैक दोषजं किञ्चित् कुष्ठं समुपलभ्यते ।
C.S.Ci. 7/10

Due to adaptation of etiological factors there will be a continuous involvement of localised *(Sthira) doṣās* for a prolonged period *(Cirakāri)* simultaneously involving tvak *(Rasadhātu), Rakta, Māṃsa* and *Ambu* leading to their *Sammārchanā* at a particular site resulting into the development of 18 varieties of *Kuṣṭha.* Three *doṣās* and four *dhātus (Rasa, Rakta, Māṃsa & Ambu)* are considered as seven fold substances and these act as a original causative factors. *Kuṣṭha* manifest by involving all the three *doṣās* and it never manifests by involving only one *doṣa. Doṣās* brings laxity in the four *dhātus,* which favours the development of *Kuṣṭha.*

सर्वाणि कुष्ठानि सवातानि सपित्तानि सश्लेष्माणि सक्रिमीणि च भवन्ति, उत्सन्नतस्तु दोषग्रहणमभिभवात् ॥ *S.S.Ni. 5/6*

There is an involvement of *Vāta, Pitta, Śleṣma* and *Krimi* in all varieties of *Kuṣṭha* and the same can be understood by observing their predominance in the manifestation of *Kuṣṭha*.

तस्य पित्तश्लेष्माणौ प्रकुपितौ परिगृह्यानिलः प्रवृद्धस्तिर्यग्गाः सिराः सम्प्रतिपद्य समुद्धूय बाह्यं मार्गं प्रति समन्ताद्विक्षिपति यत्र यत्र च दोषो विक्षिप्तो निःसरति तत्र तत्र मण्डलानि प्रादुर्भवन्ति, एवं समुत्पन्नस्त्वचि दोषस्तत्र तत्र च परिवृद्धिं प्राप्याप्रतिक्रियमाणोऽभ्यन्तरं प्रतिपद्य धातूनभिदूषयति ॥ *S.S.Ni. 5/3*

Due to exposure to etiological factors *Pitta* and *Kapha* aggravate initially followed by *Vāta*. All the increased *Doṣās* enter blood vessels and move inside in haphazard direction and reach the interior pathway and manifest disease due to laxity in *Dhātus (Rasa, Rakta, Māṁsa & Ambu)* in that particular area. Wherever these *Doṣās* move, there they manifest patches over the skin and get localised in that area. If neglected, enters into the consequent *Dhātus*.

कालेनोपेक्षितं यस्मात्सर्वं कुष्णाति तद्वपुः ।
प्रपद्य धातुन्व्याप्यान्तः सर्वान् संक्लेद्य चावहेत् ।
स स्वेदक्लेद सङ्कोथान् कृमीन् सूक्ष्मान् सुदारुणात् ।
लोमत्वक्स्नायु धमनीतरुणास्थीनि यैः क्रमात् ।
A.H.Ni. 14/4-5

As the disease advances it spreads to entire body resulting into ugly look because it brings deformity in various tissues. It is due to development of moisture inside the *Dhātus* leading to flow of sweat, exudation, putrefaction, favours the growth of minute parasites, which in turn gives trouble to body and eat away the hairs, skin, tendons, arteries, veins and cartilages one after the other.

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Pitttādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Agitated *doṣās* brings looseness in *Dhātus [Tvak (Rasa), Rakta, Māmsa* and *Ambu]*
↓
Localised in the particular area and develop morbidity in the skin
↓
Kuṣṭha (18 varieties)

**Kuṣṭha* manifest by involving all the three *doṣās* and it never manifests by involving only one *doṣa*.

Pathogenesis of dhātugata Kuṣṭa

If *Kuṣṭa* is neglected, enters into the consequent *Dhātus.*
↓
As the disease advances it spreads to entire body resulting into ugly look because it brings deformity in various tissues.
↓
It favours the development of moisture inside the *Dhātus* leading to flow of sweat, exudation, putrefaction
↓
favours the growth of minute parasites
↓
These gives troubles to body
↓
Destroy the hairs, skin, tendons, arteries, veins and cartilages one after the other.
↓
Dhātugata Kuṣṭha

**Kuṣṭha* manifest by involving all the three *doṣās* and it never manifests by involving only one *doṣa*.

कुष्ठ संप्रप्ति घटक (*Saṁprāpti Ghaṭaka of Kuṣṭha*)

Doṣa → *Pitta-Śleṣma-Vāta* in succesive order.

कुष्ठस्य पुनरमन्दपित्तमिति । *S.N. 5/3*

Involvement of *Pitta* is in the state of *Manda* (sluggish activities) [*Nyāya Candrikā]*.

Dūṣya : *Dhātu* → *Rasa, Rakta, Māṁsa* and *Lasikā* initially later all the seven *Dhātus.*

Upadhātu → *Sirā, Tvacā, Snāyu*

Other → *Ambu, Dhamanī, Taruṇāsthi*

Sārīrikamala →

Dhātumala → *Sveda, Loma, Tvacā Snehāmśa.*

Agni : *Jaṭharāgni, Dhātvāgnī*
Agnidușți : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Rasavaha, Raktavaha, Māmsavaha, Svedavaha*
Later all the remaining *Dhātuvaha Srotas* are involved.
Srotodușți : *Saṅga*
Sancārasthāna : *Sirā, Dhamani, Tiryak Sirā*
Vyaktasthāna : *Tvacā*
Udbhavasthāna : *Śākhā (Tvakādi Dhātus)*
Adhișțhāna : *Tvak, Lasikā, Asṛk*
Svabhāva : *Cirakāri*
Prabhāva : *Nava* and *Ekadoṣaja predominance Curable*
Due to negligency in treatment disease become incurable
Rasagata, Raktagata, Māmsagata→ *Curable*
Medagata → *Manageable*
Asthigata, Majjāgata & Śukragata→ Incurable
Roga bheda : *Mahākuṣṭha* → 7
Kṣudrakuṣṭha → 11

पूर्वरूप (*Prodromal Symptoms*)

स्पर्शाज्ञत्वमति स्वेदो न वा वैवर्ण्यमुन्नतिः ।
कोठानां लोमहर्षश्च कण्डूस्तोदः श्रमः क्लमः ॥
व्रणानामधिकं शूलं शीघ्रोत्पत्तिश्चिरस्थितिः ।
दाहः सुप्ताङ्गता चेति कुष्ठक्षणमग्रजम् ॥
C.S.Ci. 7/11-12

तस्य पूर्वरूपाणि त्वक् पारुष्यमकस्माद्रोमहर्षः । कण्डूः स्वेदबाहुल्यमस्वेदनं वाऽङ्गप्रदेशानां स्वापः क्षतविसर्पणमसृजः कृष्णता चेति । *S.S.Ni. 5/4*

तेषामिमानि पूर्वरुपाणि भवन्ति; तद्यथा-अस्वेदनमतिस्वेदनं पारुष्यमतिश्लक्षणता वैवर्ण्यं कण्डूर्निस्तोदः सुप्तता परिदाहः परिहर्षो लोमहर्षः खरत्वमूष्मायणं गौरवं श्वयथुर्वीसर्पागमनमभीक्ष्णं च काये कायच्छिद्रेषूपदेहः पक्व दग्ध दष्टभग्नक्षतोपस्खलितेष्वतिमात्रं वेदना स्वल्पानामपि च व्रणानां दुष्टिरसंरोहणं चेति ॥ *C.S.Ni. 5/7*

Before the actual manifestation of *Kușțha* certain Symptomatology has been observed are as follows-

- Loss of touch sensation or numbness
- Excessive perspiration or absence of perspiration
- Discolouration and appearance of rashes in the skin
- Horripilation, itching, piercing pain, physical exhaustion and tingling sensation.
- Feeling of exhaustion without any physical work followed by not suffering from difficulty in breathing and it disturbs sense perception greatly.
- Excessive pain in case of suppuration, burn, bite, fracture and dislocation.
- Excessive pain in ulcerated wounds with their quick origin and delayed healing of even small wounds.
- Production of heat and heaviness.
- Burning sensation and numbness in the extremities.
- Roughness in skin or smoothness in skin.
- Spreading of wound to other body parts and frequent occurence of oedema.
- Blackish discolouration of the blood.
- Sticking of excreta in the body especially in the orifices.

रूप (Rupa)

General Symptoms of *Kușțha* : Complete manifestation of premonitory symptoms may be considered as general characteristic features.

वात प्रधान कुष्ठ लक्षण (Vāta Pradhāna Kuṣṭha Lakṣaṇa)

कुष्ठेषु तु त्वक् संकोचस्वापस्वेदशोफभेद कौण्य स्वरोपघाता वातेन । *S.S.Ni. 5/18*

The following clinical features manifest if *Kuṣṭha* manifest due to *Vāta* Predominance : Constriction in skin, loss of sensation, sweating, swelling, cracking, shortening of extremities and loss of voice.

पित्त प्रधान कुष्ठ लक्षण (*Pitta Pradhāna Kuṣṭha Lakṣaṇa*)

पाकावदरणाङ्गुलिपतनकर्णनासाभङ्गाक्षिराग सत्वोत्पत्तयः पित्तेन। *S.S.Ni. 5/8*

The following clinical features manifest in case of *Pitta* predominant *Kuṣṭha* i:e ulceration and suppuration; cracking; loss of extremities, toes, ear and nose; redness in eye and appearance of *Krimi* over *Kuṣṭha* lesion.

कफ प्रधान कुष्ठ लक्षण (*Kapha Pradhāna Kuṣṭha Lakṣaṇa*)

कण्डूवर्ण भेद शोफस्राव गौरवाणि श्लेष्मणा। *S.S.Ni. 5/18*

The following clinical features manifest in case of *Kapha* predominant *Kuṣṭha* i:e itching, discolouration, swelling, exudation and heaviness.

Specific Symptomatology in Kuṣṭha

Classification of Kuṣṭha

तत्र सप्त महाकुष्ठानि, एकादश क्षुद्रकुष्ठानि; एवमष्टादश कुष्ठानि भवन्ति। तत्र महाकुष्ठान्यरुणौदुम्बरर्ष्यजिह्वकपालकाकणकपुण्डरीकदद्रुकुष्ठानीति। क्षुद्रकुष्ठान्यपि स्थूलारुष्कं महाकुष्ठमेककुष्ठं चर्मदलं विसर्पः परिसर्पः सिध्मं विचर्चिका किटिभं पामा रकसा चेति॥ *S.S.Ni. 5/5*

18 types of *Kuṣṭha* are described are as follows:-

Mahā Kuṣṭha-07

Kṣudra Kuṣṭha-11

Mahā Kuṣṭha-07 (*Suśruta*)	*Caraka*
1. *Aruṇa*	1. *Kāpāla*
2. *Udumbara*	2. *Audumbara*
3. *Ṛṣyajihvā*	3. *Maṇḍala*
4. *Kapāla*	4. *Ṛṣyajihva*
5. *Kākaṇaka*	5. *Puṇḍarika*
6. *Puṇḍarika*	6. *Sidhma*
7. *Dadru*	7. *Kākaṇaka*

Kṣudrakuṣṭha-11 (*Suśruta*)	*Caraka*
1. *Sthulāruṣka*	1. *Ekakuṣṭha*
2. *Mahākuṣṭha*	2. *Carmākhya*
3. *Eka Kuṣṭha*	3. *Kiṭibha*
4. *Carmadala*	4. *Vipādikā*
5. *Visarpa*	5. *Alasaka*
6. *Parisarpa*	6. *Dadru*
7. *Sidhma*	7. *Carmadala*
8. *Vicarcikā*	8. *Pāmā*
9. *Kiṭibha*	9. *Viṣphoṭaka*
10. *Pāmā*	10. *Śatāru*
11. *Rakasā*	11. *Vicarcikā*

सर्वाणि कुष्ठानि सवातानि सपित्तानि सश्लेष्माणि सक्रिमिणि च भवन्ति, उत्सन्नतस्तु दोषग्रहणमभिभवात्। *S.S.Ni. 5/6*

All kinds of *Kuṣṭa* manifest either by *Vāta*, or *Pitta* or *Śleṣma* or *Kṛimi* or in combination with *Vāta-Pitta, Kapha-Pitta* and *Vāta Kapha* or by all the three *Doṣās*.

वातेऽधिकतरे कुष्ठं कापालं मण्डलं कफे।
पित्ते त्वौदुम्बरं विद्यात् काकणं तु त्रिदोषजम्॥
वातपित्ते श्लेष्मपित्ते वातश्लेष्मणि चाधिके।
ऋष्यजिह्वं पुण्डरीकं सिध्मकुष्ठं च जायते॥
चर्माख्यमेककुष्ठं च किटिभं सविपादिकम्।
कुष्ठं चालसकं ज्ञेयं प्रायो वातकफाधिकम्॥
पामा शतारुर्विस्फोटं दद्रुश्चर्मदलं तथा।
पित्तश्लेष्माधिकं प्रायः कफप्राया विचर्चिका॥
C.S.Ci. 7/27-30

अरुः ससिध्मं रकसा महच्च यच्चैककुष्ठं कफजान्यमूनि।
वायोः प्रकोपात्परिसर्पमेकं शेषाणि पित्तप्रभवाणि विद्यात्॥
S.S.Ni. 5/16

Name of the *Kuṣṭha*	*Doṣa Predominance*
Kāpāla	→ *Vāta*
Maṇḍala	→ *Kapha*
Audumbara	→ *Pitta*
Kākaṇa	→ All the three *doṣās*
Ṛsyajihvā	→ *Vāta-Pitta*
Puṇḍarika	→ *Kapha-Pitta*
Sidhma	→ *Vāta-Kapha*
Carmākhya	→ *Vāta-Kapha*
Ekakuṣṭha	
Kiṭibha	
Vipādikā	
Alasaka	
Pāmā	→ *Pitta-Kapha*
Śatāru	
Viṣphota	
Dadru	
Carmadala	

According to *Suśruta Saṁhitā*

Name of *Kuṣṭha*		*Doṣa Predominance*
Aruṇa	→	*Vāta*
Parisarpa	→	*Vāta*
Puṇḍarika	→	*Kapha*
Audumbara	→	*Pitta*
Kapāla Kuṣṭha	→	*Pitta*
Kākaṇaka	→	*Pitta*
Visarpa	→	*Pitta*
Vicarcikā	→	*Pitta*
Kiṭibha	→	*Pitta*
Pāmā	→	*Pitta*
Rṣyajihva	→	*Pitta*

सर्वं त्रिदोषजं कुष्ठं दोषाणां तु बलाबलम्।
C.S.Ci. 7/31

All varieties of *Kuṣṭha* manifest due to all the three *Doṣās* but expression of symptoms may be by a dominated *Doṣās.*

Specific Symptoms of *Kuṣṭha*

कापाल कुष्ठ (*Kāpāla Kuṣṭha*)

कृष्णारुणकपालाभं यद्रूक्षं परुषं तनु।
कापालं तोदबहुलं तत्कुष्ठं विषमं स्मृतम्॥
C.Ci. 7/14

कृष्णकपालिकाप्रकाशानि कपालकुष्ठानि।
S.Ni. 5/8

Characteristic features of *Kāpāla Kuṣṭha* are as follows-

- Blackish-red patches resemble like that of broken pieces of earthen pot.
- Patches are rough, coarse and thin to touch with severe pain.
- It is incurable.

औदुम्बर (*Audumbara*)

दाहकण्डूरुजारागपरीतं लोमपिञ्जरम्।
उदुम्बरफलाभासं कुष्ठमौदुम्बरं विदुः॥
C.Ci. 7/15

पित्तेन पक्वोदुम्बरफलाकृतिवर्णान्यौदुम्बराणि।
S.Ni. 5/8

It is diagnosed based on following characteristics:-

- Burning sensation, itching, pain and redness in the affected area.
- Colour of the hair in the affected area changed to brown in colour.
- Whole appearance of the symptoms resemble like that of ripened *Udumbara* fruit (fig) especially in respect to shape & colour.
- It manifests due to abnormal *Pitta.*

मण्डल कुष्ठ (*Maṇḍala Kuṣṭha*)

श्वेतं रक्तं स्थिरं स्त्यानं स्निग्धमुत्सन्नमण्डलम्।
कृच्छ्रमन्योन्यसंसक्तं कुष्ठं मण्डलमुच्यते॥
C.Ci. 7/16

Clinical features of *Maṇḍala Kuṣṭha* are as follows :

- Patches are white and red in colour.
- Patches are stable, compact, unctuous and circular in shape with elevated margins.
- It is difficult for management.
- Patches are matted with each other.

ऋष्यजिह्व (*Ṛsyajihva*)

कर्कशं रक्तपर्यन्तमन्तःश्यावं सवेदनम्।
यदृष्यजिह्वासंस्थानमृष्यजिह्वं तदुच्यते॥
C.S.Ci. 7/17

ऋष्यजिह्वाप्रकाशानि खराणि ऋष्यजिह्वानि।
S.S.Ni. 5/8

It is diagnosed based on following characteristic features:-

- Patches are rough with red margins.
- Patches are internally blackish or brownish discolouration.
- Patches are painful
- Appearance of the symptomatology similar to that of tongue of *Ṛsya* (a type of antelope with blue testicles).

पुण्डरीक कुष्ठ (*Puṇḍarika Kuṣṭha*)

सश्वेतं रक्तपर्यन्तं पुण्डरीकदलोपमम्।
सोत्सेधं च सदाहं च पुण्डरीकं तदुच्यते॥
C.S.Ci. 7/18

पुण्डरीकपत्रप्रकाशानि पौण्डरीकाणि श्लेष्मणा।
S.S.Ni. 5/8

It is diagnosed by following clinical features:-

- Patches are raised with red margins having whitish discolouration resemble like the leaf of lotus.

- Burning sensation

सिध्म कुष्ठ (Sidhma Kuṣṭha)

श्वेतं ताम्रं तनु च यद्रजो घृष्टं विमुञ्चति।
अलावूपुष्पवर्णं तत् सिध्मं प्रायेण चोरसि॥
C.S.Ci. 7/19

कण्ड्वन्वितं श्वेतमपायि सिध्म विद्यात्तनु प्रायश ऊर्ध्वकाये।
S.S.Ni. 5/12

It is diagnosed by following clinical features:-

- Patches are white and copper colour.
- Patches are thin, leaves out dust like powder on rubbing, which is in the form of dust.
- The whole appearance of disease simulating like that of *alābupuṣpa* (flower of bottle gourd). Main location is chest.
- Itching in the affected parts.
- Generally seen in the upper part of the body and usually not troublesome.

Suśruta included this diseases under *Kṣudra Kuṣṭha.*

काकण कुष्ठ (Kākaṇa Kuṣṭha)

यत् काकणन्तिकावर्णमपाकं तीव्रवेदनम्।
त्रिदोषलिङ्गं तत् कुष्ठं काकणं नैव सिध्यति॥
C.S.Ci. 7/20

काकणन्तिकाफलसदृशान्यतीव रक्तकृष्णानि काकणकानि।
S.S.Ni. 5/8

It can be diagnosed by following clinical features:-

- Patches are red in colour simulates like that of *Guñjā* seed (*Abrus precatorius Linn.*)
- Patches does not suppurate and manifest severe pain.
- Appearance of all the symptoms of *Tridoṣa* variation.
- It is incurable. Some patches are black in colour.

Common Symptoms are found in *Audumbara, Ṛṣyajihvā, Kāpāla* and *Kākaṇañtika* in addition to the above specific symptoms mentioned earlier are as follows:-

तेषां चतुर्णामप्योषचोषपरिदाहधूमायनानि क्षिप्रोत्थानप्रपाकभेदित्वानि क्रिमिजन्म च सामान्यानि लिङ्गानि।

S.S.Ni. 5/8

- Burning sensation in the affected part as well as in whole body.
- Sucking type of pain
- Patient feels as if hot fumes are coming out from mouth
- Onset, suppuration, progress and development of the *Krimi* is quick.

अरुण कुष्ठ (Aruṇa Kuṣṭha)

तत्र, वातेनारुणाभानि तनूनि विसर्पीणि तोदभेदस्वापयुक्तान्यरुणानि। *S.S.Ni. 5/8*

It is diagnosed by following clinical features:-

- Patches are light red in colour, thin and spreads to different body parts.
- Nature of the pain is either pricking or cutting.
- Loss of sensation in affected areas.

  It is included under *Mahākuṣṭha* in *Suśruta Saṁhitā.*

दद्रु कुष्ठ (*Dadru Kuṣṭha*)

अतसीपुष्पवर्णानि ताम्राणि वा विसर्पीणि पिडकावन्ति च दद्रुकुष्ठानि। *S.S.Ni. 5/8*

सकण्डूरागपिडकं दद्रुमण्डलमुद्गतम्॥ *C.S.Ci. 7/23*

It is diagnosed by following features:-

- Patches are coppery in colour
- Spreads quickly by manifesting skin eruptions.
- Patches are charcterised by itching, red in colour with development of pimples and circular in shape.
- Symptomatology of the disease appears like the flower of *Atasi* (i:e red in colour)

  It is included under *Mahākuṣṭha* and *Kṣudrakuṣṭha* in *Suśruta Saṁhitā* & *Caraka Saṁhitā* respectively.

General clinical features observed in *Puṇḍarika* and *Dadru Kuṣṭha* in addition to the specific symptomatology mentioned above are as follows:-

तयोर्द्वयोरप्युत्सन्नता परिमण्डलता कण्डूश्चिरोत्थानत्वं चेति सामान्यानि रूपाणि। *S.S.Ni. 5/8*

- Patches are elevated and circular in shape.
- Itching and onset of the disease is slow.

क्षुद्र कुष्ठ (Kṣudra Kuṣṭha)

एक कुष्ठ (*Eka Kuṣṭha*)

अस्वेदनं महावास्तु यन्मत्स्यशकलोपमम् ।
तदेककुष्ठं, ... ॥

C.S.Ci. 7/21

कृष्णारुणं येन भवेच्छरीरं तदेककुष्ठं प्रवदन्ति कुष्ठम् ।

S.S.Ni. 5/10

It is diagnosed by observing following clinical features:-

- Absence of perspiration, occupies larger areas and simulates like fish scales.
- Entire body becomes blackish red.

चर्माख्यं (*Carmākhyam*)

चर्माख्यं बहलं हस्तिचर्मवत् । *C.S.Ci. 7/21*

Affected area becomes thick like elephant skin is diagnosed as *Carmākhya.*

किटिभ कुष्ठ (*Kiṭibha Kuṣṭha*)

श्यावं किणखरस्पर्शं परुषं किटिभं स्मृतम् ।

C.S.Ci. 7/21

यत् स्रावि वृत्तं घनमुग्रकण्डु तत् स्निग्धकृष्णं किटिभं वदन्ति ।

S.S.Ni. 5/14

It is diagnosed by observing following clinical features : Patches are blackish brown in clour, rough like a corn and coarse in nature. Patches are exudative, round, thick. Patches are unctuous and black with severe itching.

विपादिका कुष्ठ (*Vipādikā Kuṣṭha*)

कण्डूमती दाहरुजोपपन्ना विपादिका पादगतेयमेव ॥

S.S.Ni. 5/13

वैपादिकं पाणिपादस्फुटनं तीव्रवेदनम् ॥

C.S.Ci. 7/22

It is diagnosed by following clinical features : Manifestation of cracks in hand & feet with excessive pain associated with itching and burning sensation.

अलसक कुष्ठ (*Alasaka Kuṣṭha*)

कण्डूमद्भिः सरागैश्च गण्डैरलसकं चितम् ।

C.S.Ci. 7/23

It is diagnosed by observing following clinical features : Appearance of nodular growth associated with itching and reddish discolouration.

दद्रु कुष्ठ (*Dadru Kuṣṭha*) *C.S.Ci. 7/23*

Discussed under *Mahākuṣṭha* because *Suśruta* considered in that category.

चर्मदल कुष्ठ (*Carmadala Kuṣṭha*)

रक्तं सकण्डू सस्फोटं सरुग्दलति चापि यत् ।
तच्चर्मदलमाख्यातं संस्पर्शासहमुच्यते ॥

C.S.Ci. 7/24

स्युर्येन कण्डूव्यथनौषचोषास्तलेषु तच्चर्मदलं वदन्ति ॥

S.S.Ni. 5/10

It is diagnosed by observing following features : Patches are reddish associated with itching, pain, burning sensation and sucking type of pain in the palms and soles.

पामा (*Pāmā*)

पामा श्वेतारुणश्यावाः कण्डूलाः पिण्डका भृषम् ।

C.S.Ci. 7/25

सास्त्रावकण्डूपरिदाहकाभिः पामाऽणुकाभिः पिडकाभि र्ह्या ।
(स्कोटैः सदाहैरति सैव कृच्छः स्फिक्पाणिपादप्रभवैर्निरुहया) ॥

C.S.Ci. 7/14-25

It is diagnosed by following clinical features : Excessive itching, patches are either white, reddish or blackish brown in colour. Skin eruptions are small in size and exudates comes out from them and burning sensation all over the body.

कच्छु (*Kacchu*)

Development of visicles associated with burning sensation and appear on the buttock, hands and feet are known as *Kacchū.*

स्फोट (*Sphoṭa*)

स्फोटाः श्वेतारुणाभासो विस्फोटाः स्युस्तनुत्वचः ।

C.S.Ci. 7/25

It is diagnosed by following clinical features : Skin eruptions are either white or reddish having thin skin.

शतारु (*Śatāru*)

रक्तं श्यावं सदाहार्ति शतारुः स्याद्बहुव्रणम् ।
C.S.Ci. 7/26

It is diagnosed by following clinical features : Multiple skin eruptions with ulceration and the colour is red or blackish brown in colour and it is associated with burning sensation and pain.

विचर्चिका (*Vicarcikā*)

सकण्डूः पिडका श्यावा बहुस्त्रावा विचर्चिका ॥
C.S.Ci. 7/26

राज्योऽतिकण्ड्वर्त्तिरुजः सरूक्षा भवन्ति गात्रेषु विचर्चिकायाम् ।
S.S.Ni. 5/13

It is diagnosed by following clinical features : Skin eruptions are black in colour having excessive exudation and itching; striae appear all over the body which are dry, painful along with itching.

Description of Mahākuṣṭha in Caraka Samhitā

कपाल कुष्ठ (*Kapāla Kuṣṭha*)

रूक्षारुणपरुषाणि विषमविसृतानि खरपर्यन्तानि तनून्युद्धतवहिस्तनूनि सुप्तवत्सुप्तानि हृषितलोमाचितानि निस्तोदवहुलान्यल्पकण्डूदाहपूयलसीकान्याशुगतिसमुत्थानान्याशुभेदीनि जन्तुमन्ति कृष्णारुणकपालवर्णानि च कपालकुष्ठानीति विद्यात् । *C.S.Ni. 5/7 (1)*

- Patches are dry (rough), reddish, coarse, uneven shape, having coarse edges which is thin and elevate in the periphery.
- Excessive numbness.
- Area is covered by erect hairs associated with severe pain. Mild itching, burning sensation, discharge of pus along with serous exudation.
- Development, causation and ulcerations are instanstaneous.
- Appearance of parasites and resemble like pieces of earthen pot having black or reddish colour.

औदुम्बर कुष्ठ (*Audumbara Kuṣṭha*)

ताम्राणि ताम्रखररोमराजीभिरवनद्धानि बहलानि बहुबहलपूयरक्तलसीकानि कण्डूक्लेदकोथदाहापाकवन्त्याशुगतिसमुत्थानभेदीनि ससंतापक्रिमीणि पक्वोदुम्बरफलवर्णान्यौदुम्बरकुष्ठानीति विद्यात् ।*C.S.Ni. 5/7 (2)*

- Patches are coppery in colour surrounded by coppery discoloured hairs.
- Patches are thick and associated with pus, blood and serous exudation in large quantity.
- Patches are associated with itching, sticky exudation, sloughing, burning sensation and suppuration.
- Causation, manifestation and ulceration are very fast.
- Increased temperature associated with parasitic infestations.
- Appearance of the whole disease resemble like ripened *Udumbara* fruit.

मण्डल कुष्ठ (*Maṇḍala Kuṣṭha*)

स्निग्धानि गुरूण्युत्सेधवन्ति श्लक्ष्णस्थिरपीतपर्यन्तानि शुक्लरक्तावभासानि शुक्लरोमराजीसन्तानानि बहुबहलशुक्लपिच्छिलस्त्रावीणि बहुक्लेदकण्डूक्रिमीणि सक्तगतिसमुत्थानभेदीनि परिमण्डलानि मण्डलकुष्ठानि विद्यात् ।
C.S.Ni. 5/7 (3)

Patches are unctuous, heavy, swollen associated with fixed and yellow border. These are either white or red in colour associated with white hairs in large quantity. Excessive thick exudation having white colour associated with itching and copious parasitic infestations.

Causation, manifestation and ulceration are sluggish and resemble like round in shape.

ऋष्यजिह्वा (*Ṛṣyajihvā*)

पुरुषाण्यरुणवर्णानि बहिरन्तः श्यावानि नीलपीतताम्रावभासान्याशुगतिसमुत्थानान्यल्पकण्डूक्लेदक्रिमीणि दाहभेदनिस्तोद (पाक) बहुलानि शूकोपहतोपमवेदनान्युत्सन्नमध्यानि तनुपर्यन्तानि कर्कशपिडकाचितानि दीर्घपरिमण्डलान्यृष्यजिह्वाकृतीनि ऋष्यजिह्वानीति विद्यात् । *C.S.Ni. 5/7 (4)*

Patches are rough, reddish in colour having blackish discolouration at the interior and exterior lesions. Lustre or shade of the lesion is either blue, yellow or coppery colour.

Causation and manifestations are instantaneous. Itching, oozing and parasitic infestations are mild. But it is associated with excessive burning sensation and pain i:e cutting & piercing pain along with suppuration.

Severe pain as if inflicted with bristles. In the centre, *Patches* are elevated patches and their margins are thin. It is associated with coarse boils, having large circumference and resemble like the tongue of while footed antelpe (deer).

पुण्डरीक कुष्ठ (*Puṇḍarīka Kuṣṭha*)

शुक्लरक्तावभासानि रक्तपर्यन्तानि रक्तराजीसिरासन्तान्युत्सेधवन्ति बहुबहलरक्तपूयलसीकानि कण्डूक्रिमिदाहपाकवन्त्याशुगतिसमुत्थानभेदीनि पुण्डरीकपलाशसंकाशानि पुण्डरीकाणीति विद्यात् ।*C.S.Ni. 5/7 (5)*

Luster or shade of the patches is either white or red. Having red margins pervaded with red blood vessels.

Copious discharge of thick blood, pus and serous associated with itching, parasitic infestations, burning sensation and suppuration. Causation, manifestation and ulcerations are instantaneous. Appearance of disease resembles like that of petals of lotus & flower.

सिध्म कुष्ठ (*Sidhma Kuṣṭha*)

परुषारुणानि विशीर्णबहिस्तनून्यन्तः स्निग्धानि शुक्लरक्तावभासानि बहून्यल्पवेदनान्यल्पकण्डूदाहपूयलसीकानि लघुसमुत्थानान्यल्पभेदक्रिमीण्यलाबुपुष्पसङ्काशानि सिध्मकुष्ठानीति विद्यात् । *C.S.Ni. 5/7 (6)*

Patches are rough and reddish in colour. Periphery is fissured and thin. Central portion is smooth. Shade is either white or red associated with numerous number having little pain, itching, burning sensation, pus and serous discharge.

Causation and ulcerating are sluggish with less parasitic infestation. Appearance of the disease resembles like bottle gourd flower.

काकण कुष्ठ (*Kākaṇa Kuṣṭha*)

काकणन्तिकावर्णान्यादौ पश्चातु सर्वकुष्ठलिङ्गसमन्वितानि पापीयसा सर्वकुष्ठलिङ्गसंभवेनानेकवर्णानिकाकणानीति विद्यात्। तान्यसाध्यानि, साध्यानि पुनरितराणि॥

C.S.Ni. 5/8

Patches are red in colour like that of *Gunjā* seeds. In the initial stage but later it is associated with appearances of all symptoms of sinful *Kuṣṭhās.* That's why, later it develops multiple colour in the lesions. It is incurable.

धातुगत कुष्ठ (*Dhātugata Kuṣṭha*)

रस धातुगत कुष्ठ (*Rasa Dhātugata Kuṣṭha*)

स्पर्शहानिः स्वेदनत्वमीषत्कण्डूश्च जायते ।
वैवर्ण्यं रूक्षभावश्च कुष्ठे त्वचि समाश्रिते ॥

S.S.Ni. 5/22

***Rasadhātugata Kuṣṭha* is diagnosed by observing following clinical features :**

- Loss of touch sensation
- Persipiration, mild itching, discolouration, roughness.

रक्त धातुगत कुष्ठ (*Raktadhātugata Kuṣṭha*)

त्वक्स्वापो रोमहर्षश्च स्वेदस्याभिप्रवर्त्तनम् ।
कण्डूर्विपूयकश्चैव कुष्ठे शोणितसंश्रिते ॥

S.S.Ni. 5/23

***Raktadhātugata Kuṣṭha* is diagnosed by following clinical features :** Numbness in skin, horripilation, sweating, itching, formation of pus having bad smell.

मांस धातुगत कुष्ठ (*Māmsadhātugata Kuṣṭha*)

बाहुल्यं वक्त्रशोषश्च कार्कश्यं पिडकोद्गमः ।
तोदः स्फोटः स्थिरत्वं च कुष्ठे मांससमाश्रिते ॥

S.S.Ni. 5/24

***Māmsadhātugata Kuṣṭha* is diagnosed by following clinical features :** Thickness of skin, dryness of the mouth, roughness of the skin, appearance of skin eruptions, pricking and bursting pain and stability.

मेद धातुगत कुष्ठ (*Medadhātugata Kuṣṭha*)

दौर्गन्ध्यमुपदेहश्च पूयोऽथ क्रिमयस्तथा ।
गात्राणां भेदनं चापि कुष्ठे मेदःसमाश्रिते ॥

S.S.Ni. 5/25

***Medadhātugata Kuṣṭha* is diagnosed by observing following clinical features :** Bad smell and accumulation of dirt over body, pus formation, manifestation of parasitic infestations *(Krimi)* in the wounds and mutilation of body parts.

अस्थिमज्जागत कुष्ठ *(Asthi Majjāgata Kuṣṭha)*

नासाभङ्गोऽक्षिरागश्च क्षते च क्रिमिसंभवः।
भवेत् स्वरोपधातश्च ह्यास्थिमज्जसमाश्रिते॥

S.S.Ni. 5/26

***Asthi-Majjāgata Kuṣṭha* is diagnosed by following clinical features :** Deformity in nose, reddish discolouration of eyes, appearance of parasitic infestations in the wounds and loss of voice.

शुक्रधातुगत कुष्ठ *(Śukradhātugata Kuṣṭha)*

कौण्यं गतिक्षयोऽङ्गानां संभेदः क्षतसर्पणम्।
शुक्रस्थानगते लिङ्गं प्रागुक्तानि तथैव च॥

S.S.Ni. 5/27

***Śukradhātugata Kuṣṭha* is diagnosed by following clinical features :** Shortening of extremities, limited motion, splitting of body parts, gradual spreading of wounds and all above symptoms.

उपद्रव *(Upadrava)*

एवं कुष्ठं समुत्पन्नं त्वचि कालप्रकर्षतः।
क्रमेण धातून् व्याप्नोति नरस्याप्रतिकारिणः॥

S.S.Ni. 5/21

Initially *Kuṣṭha* is confined to skin and gradually invades the other tissues in due course of time, if it is not treated early and properly.

साध्यानामपि ह्युपेक्ष्यमाणानां त्वङ्मांसशोणितलसीकाकोथक्लेदसंस्वेदजाः क्रिमयोऽमूर्च्छन्ति; ते भक्षयन्तस्त्वगादीन् दोषाः पुनर्दूषयन्त इमानुपद्रवान् पृथक् पृथगुत्पादयन्तितत्र वातः श्यावारुणवर्णं परुषतामपि च रौक्ष्यशूलशोषतोदवेपथुहर्षसङ्कोचायासस्तम्भसुप्तिभेदभङ्गान्, पित्तं दाहस्वेदक्लेदकोथस्त्रावपाकरागान्, श्लेष्मा त्वस्य श्वैत्यशैत्यकण्डूस्थैर्यगौरवोत्सेधोपस्नेहोपलेपान्, क्रिमयस्तु त्वगादींश्चतुरः सिराः स्नायूश्चास्थीन्यपि च तरुणान्याददते॥

अस्यां चैवावस्थायामुपद्रवाः कुष्ठिनं स्पृशन्ति; तद्यथा-प्रस्त्रवणम ङ्गभेदः पतनान्यङ्गावयवानां तृष्णाज्वरातीसारदाहदौर्बल्यारोचकाविपाकाश्च, तथाविधमसाध्यं विद्यादिति॥

C.S.Ni. 5/10-11

If treatment is not employed correctly & early leads to development of *Krimi* over *Tvaca, Māmsa, Rakta* and *Lasika, Kotha* (slough), *Kleda* (discharge) and *Sveda* (sweat). *Krimi* gradually invaded to deeper *Dhātus* and start eating other tissues & softening the body parts.

Doṣaja Upadrava manifest in Kuṣṭha are as follows

***Upadrava* due to *Vāta* predominant *Kuṣṭha* are as follows :** Blackish brown and reddish colour, roughness, dryness, piercing pain, emaciation, pricking pain, trembling, horripilation, contraction, exhaustion. Stiffness, numbness, decay and breaking down of body parts.

***Upadrava* due to *Pitta* predominant *Kuṣṭha* are as follows :** Burning sensation, sweating, moistening, sloughing, serous discharge, suppuration and redness.

***Upadrava* due to *Kapha* predominant *Kuṣṭha* are as follows :** Whitishess, coldishness, itching, steadiness, heaviness, swelling, unctousness and adhesion. Krimi invades to *Tvak, Rakta, Māmṣa, Lasikā, Sirā, Snāyu, Asthi, Taruṇāsthi.* As a result following complications manifest. Profuse exudation, ulceration of body parts, sequestration of the organs of the body, thirst, fever, diarrhoea, burning sensation, general debility, anorexia and indigestion. *Kuṣṭha* with such type of complications are incurable.

साध्यासाध्यता *(Sādhyāsādhyatā)*

सवैलिङ्गैर्यक्तं मतिमान् विवर्जयेदबलम्।
तृष्णादाहपरीतं शान्ताग्निं जन्तुभिर्जग्धम्।
वातकप्रबल याधोदेकदोषोल्बणं न तत् कृच्छ्रम्।
कफपित्त वातपित्तप्रबलानि तु कृच्छ्रसाध्यानि।

C.S.Ci. 7/37-38

Don't treat *Kuṣṭha* patients if following symptoms are observed;

Symptomatology of all the *Tridoṣaja* features, debility, severe thirst, burning sensation, complete loss of digestive fire and development of *Krimi* over the affected lesions. *Vāta-Kapha* predominant and single *Doṣa* predominant is not difficult for management. However predominance of *Kapha-Pitta* or *Vāta-Pitta* are difficult for management.

तत्र यदसाध्यं तदसाध्यतां नातिवर्तते, साध्यं पुनः किंचित् साध्यातामतिवर्तते कदाचिदपचारात्। *C.Ni. 5/9*

Incurable *Kuṣṭhā's* are always incurable, however, curable *Kuṣṭhās* may become incurable due to unwholesome regimen.

उपशय/पथ्य (*Upaśaya/Pathya*)

C.Ci. 7th Chapter, *S.Ci.* 9th Chapter, *B.P.* 54th Chapter, *A.H.Ci.* 19th Chapter

Auṣadha → कुष्ठे खदिरः *C.Ni. 3/10 (Gaṅgādhara)* *Khadira* is best drug for *Kuṣṭha*, *Rasona, Haritaki, Pippali, Kuṣṭhaghna Mahā-kaṣāya, Āragvadhādi gana, Sālasarādi gaṇa, Lākṣādi gaṇa, Paṭolādi Kasāya.*

Cūrṇa → *Mustādi Cūrṇa, Triphalādi Cūrṇa, Kuṣṭādi Cūrṇa, Hapuṣādi Cūrṇa. Lauha Rasayana, Ayaṣkriti.*

Āsava → *Lodhrāsava, Madhvāsava, Triphalāsava.*

Ariṣta → *Kanakabindvāriṣṭa, Khadirāriṣṭa, Rohit-akāriṣṭa*

Vaṭi → *Pathyādi Guṭikā, Maṇḍūra Vaṭaka, Candraprabhāvaṭi.*

Guggulu → *Kaiśora Guggulu, Mahāyogaraja Guggulu, Eka Viṁśatika, Guggulu.*

Yavāgu → *Khadira Yavāgu*

Rasa Auṣadāhi → *Tālakeśvara Rasa, Kuṣṭha kuṭhāra rasa, Mahā tālakeśvara Rasa.*

Avaleha → *Danti haritaki, Amrita Bhallatakāvaleha, Maha Bhallātakāvaleha.*

Ghṛta → *Nīlinādhya Ghṛta, Paṭola Ghṛta, Mahatiktaka Ghṛta, Nimba Ghṛta, Pancatikta Ghṛta.*

Bāhya Snehana

Bhallātaka Taila, Tuvaraka Taila

Rasāyana → *Endra Rasāyana*

Lepa → *Citrakādi lepa, Kuṣṭha Nāśaka lepa, Indrayava Kalka, Sidhma lepa, Apāmārga Lepa, Guḍūcī Lepa.*

Anna → *Śāli and Ṣaṣṭika Anna (old rice), Mudga, Āḍhaki, Nimba, Flesh of Jāṅgala animals devoid of fat, Khadira decoction for drink.*

विहार (*Vihāra*)

Khadira decoction bath, *Siddhārthaka Snāna, Upavāsa, Thirst, Ātapa Sevana, Māruta Sevana, Ubaṭana* by *Cakramarda* seeds, *Sarja Rasa.*

ततः शालिषष्टिकयवगोधूमकोरदूषश्यामाकोद्दालकादीननवान् भुञ्जीत मुद्गाढक्योरन्यतरस्य यूषेण सूपेन वा निम्बपत्रारुष्कर-व्यामिश्रेण मण्डूकपर्ण्यवल्गुजाटरूषकरूपिकापुष्पैः सर्पिःसिद्धैः सर्षपतैलसिद्धैर्वा, तिक्तवर्गेण वाऽभिहितेन; मांससात्म्याय वा जाङ्गलमांसममेदस्कं वितरेत्; तैलं वज्रकमभ्यङ्गार्थे, आरग्वधादिकषायमुत्सादनार्थे, पानपरिषेकावगाहादिषु च खदिरकषायम्, इत्येष आहाराचारविभागः ॥ *S.S.Ci. 9/5*

Patients of *Kuṣṭha* should consume food prepared from old *Śāli, Ṣaṣṭika, Yava, Godhūma, Koradūṣa, Śyāmāka, Uddālaka* etc. *Yūṣa* (soup) prepared from *Mudga, Āḍhaki, Sūpa* (meat soup) added with *Nimba Patra* or *Āruṣkara Patra.*

Intake of ghee or *Sarṣapa Taila* (mustard oil) boiled with *Maṇḍūkaparṇi, Avalgujā, Āṭārūṣaka* and *Rupika Puṣpa* or with drugs of bitter taste group (*Tikta Varga*).

For non-vegetarian person meat soup of animals of *Jāṅgala* (desert land) region may be given, it should be free from fat.

Vajraka taila should be used regularly for anointing and massage.

Āragvadādi gaṇa decoction for massage, *Khadira* decoction for drinking, pouring over body and for tub bath.

In cases of simple *Kuṣṭha* one should have the *Pracchāna* therapy, but whereas it is the case of severe *Kuṣṭha* one should have the *Śirāvyadha.*

When the *Doṣās* are highly concentrated, after diagnosing the condition and strength of the patient, proper and continuous treatment (*Samśodhana - Vamana*, *Virecana*, etc.) should be administered. (B.R. 54/2)

The following measures and products are considered to be beneficial for a patient of *Kuṣṭha*: doing *Vamana* therapy after every 15 days, doing *Virecana* (purgation) therapy every month, doing nasal therapy after every three months, opening *Phasta* after every six months period, intake of *Ghṛtam*, pasting (*Lepa*), old barley, wheat, *Śāli* rice, *Mudga*, *Arahara*, *Masūra*, honey, meat of wild animals and birds, fruits (*Karkaṭī*, cucumber and the like) which grows in *Āṣāḍha* month, soft leaves of *Vetra*, *Parbara*, *Vyāghrī* fruit, *Kākamācī*, *Neem* leaves, garlic, *Sūryāvarta*, *Punarnavā*, *Meṣaśṛṅgī*, leaves of *Cakramarda*, *Bhallātaka*, ripe fruits of palm, *Khadira*, *Citraka*, *Triphalā*, *Jātīphala*, *Nāgakeśara*, *Keśara*, old *Ghṛtam*, bitter *Koṣatakī*, *Karañja*, sesame oil, mustard oil, *Neem* oil, *inguda* oil, light rice, oils of *Sarala* wood, *Devadāru*, *Śimśapa* and *Aguru*, urines of cow, donkey, camel, horse and buffalo, *Kastūrī*, *Gandhaka*, *Tikta* products and *Kṣāra* therapy. (B.R. 54/372-378)

अनुपशय/अपथ्य (*Anupaśaya/Apathya*)

S.S.Ci. 9th chapter, *A.H.Su.* 14th chapter

Meat of animal and bird residing in aquatic region, milk, curd, *Tila, Kulattha, Māṣa, Niṣpāva, Sugar & Jaggery* Preparations. Sour taste, pungent taste, salt, incompatible foods, over eating, consumption of food before the digestion of previous meal, over eating, sleeping during daytime, excessive indulgence in sex. All *Kuṣṭha Nidāna.*

तत्र त्वग्दोषी मांसवसादुग्धदधितैलकुलत्थमाषनिष्पावेक्षुपिष्टविकाराम्लविरुद्धाध्यशनाजीर्णविदाह्यभिष्यन्दीनि दिवास्वप्नं व्यवायं च परिहरेत् ॥ *S.S.Ci. 9/4*

The following regimen should be strictly avoided for *Kuṣṭha* patients : Avoid the consumption of Meat, Fat, Milk, Curd, Oil, *Kulattha, Māṣa, Niṣpāva, Ikṣu, Piṣṭa Vikāra* (eatable prepared from corn flour). Sour preparations, in compatible foods, over eating, food which is not properly processed, food which causes burning sensation, food which promotes moisture inside the tissues or blocks the channel, sleeping during day time and sexual intercourse.

In *Kuṣṭha* disorder, foods containing citric, salty and warm (*Kaṭu*) properties should be avoided. Also, curd, milk, *Guḍa*, meat of marshy animals and birds, sesame and *Urada* pulse should be avoided. (B.R. 54/371)

The following measures and products are considered to be harmful to the patient of *Kuṣṭha*: involvement in evil deeds, denying their evil deeds, disrespecting teachers, threatening teachers, eating and drinking incompatible foods, sleeping during day time, roaming under extreme heat of sun, eating unequal foods, *Svedana* therapy, sex, obstructing the flow of urine and stool, eating products of sugarcane, wresting, exercising, sour edibles, sesame, *Urada*, thin products, heavy rice, fresh rice, products that have burning sensation and *Viṣṭambhakāraka*, radish, water of rivers originated from the mountains of *Sahyādri* and *Vindhyācala*, meat of marshy animals and birds, curd, milk, liquor and raw sugar (*Guḍa*). (B.R. 54/379-381).

Differential diagnosis of Mahākuṣṭha and Kṣudra Kuṣṭha

S.No.	*Mahākuṣṭha*	*Kṣudra Kuṣṭha*
Doṣa	*Bahudoṣa*	*Alpadoṣa*
Clinical features	Many clinical presentations	less clinical presentations
Pain and discomfort	More	less
Dhātus involvement	Deeper *dhātus* involved	Superficial dhatus involved
Cikitsā	Purification followed by pacification and local application measures required	Pacification and local application measures required

Prabhāva	May be chronic	May not be chronic Some may be chronic
Deformities Morphological changes	Manifestation of deformities More morphological changes with widespread Involvement of area with chronicity and complications	May not develop deformities Less morphological changes with local spread Involvement of area with no chronicity and complications. However some *Kṣudra Kuṣṭha* may be superficially widespread over the skin with chronicity and less complications
Classification *Suśruta* S.S.Ni. 5/5	**07** 1. *Aruṇa* 2. *Audumbara* 3. *Ṛṣyajīhvā* 4. *Kapāla* 5. *Kākaṇaka* 6. *Puṇḍarīka* 7. *Dadru*	**11** 1. *Sthūlaruṣka* 2.*Mahākuṣṭha* 3. *Eka Kuṣṭha* 4. *Carmadala* 5. *Visarpa* 6. *Parisarpa* 7. *Sidhma* 8. *Vicarcikā* 9. *Kiṭibha* 10.*Pāmā* 11.*Rakaṣā*
Caraka C.S.Ci. 7/13-26	1. *Kapāla* 2. *Audumbara* 3. *Maṇḍala* 4. *Ṛṣyajīhvā* 5. *Puṇḍarīka* 6. *Sidhma* 7. *Kakaṇaka*	1. *Ekakuṣṭha* 2.*Carmākhya* 3.*Kiṭibha* 4.*Vipādika* 5.*Alasaka* 6. *Dadru* 7.*Carmadala* 8.*Pāmā* 9.*Visphoṭaka* 10. *Śataru* 11.*Vicarcika*

Note - Kāśyap Saṁhitā*-Cikitsasthāna -Kuṣṭha cikitsā Adhyāy*

Sādhya Kuṣṭha -9

1. *Sidhma* 2. *Vicarcikā* 3. *Pāmā* 4. *Dādru* 5. *Kitibha*
6. *Kapāla* 7. *Śthūlāruṣka* 8. *Maṇḍala* 9. *Viṣaja*

Asādhya Kuṣṭa -9

1. *Puṇḍarika* 2. *Śvitra* 3. *Rṣyajīhvā* 4. *Śatāruṣka* 5. *Audumbhara*
6. *Kākaṇaka* 7. *Carmadala* 8. *Ekakuṣṭha* 9. *Vipādikā*

A separate chapter on *Carmadala* has been described & 4 varieties namely *Vātaja, Pittaja, Kaphaja* and *Sannipātaja* (K.S.Khi. 15 th Chapter)

Differentiate Mahā Kuṣṭa and Kṣudra Kuṣṭa

S.N.	*Kṣudra Kuṣṭa*	*Lakṣaṇas*	Skin Disorders	Signs and Symptoms
1	*Ekakuṣṭa*	*Doṣa- Vāta Kapha* resembles *Matsyaśakalopam* (scales of fish), *Mahāvastu, Aśweda*	Icthyosis	Fish skin Dry, scaly skin Deep painful cracks
2	*Carmākya*	*Doṣa- Vāta Kapha Hasticarmavāt* (skin is thick resembling elephant skin), *Bahala*	Lichen Simplex Chronicus	Leathery induration, well circumscribed Erythematous Thickened lichenified skin
3	*Kiṭibha*	*Doṣa- Vāta Kapha Śyāva Kinakarasparśa* (touch similar to granulation process in wound), *Paruśa* (hard to touch) *Kaṇḍu*	Psoriasis	Silvery scales Erythematous Rounded plaques Roughness of skin Intense Itching

4	*Vipādikā*	*Doṣa- Vāta Kapha Panipadasphūṭana Tīvravedana*	Palmo Plantar Psoriasis	Affects palms & soles Itching, scaling Cracking, raised thickened skin
5	*Visphoṭa*	*Doṣa- Pitta Kapha Śvetāruna Tanutvaca*	Folliculitis	Small white headed pimples Red inflamed skin Pus filled sores
6	*Śataru*	*Doṣa- Pitta Kapha Rakta, Śyāva varṇa Dāha, arati Bahu varuṇa*	Furuncle	Typical red Contains pus Painful, tender
7	*Vicarcikā*	*Doṣa- Pitta Kapha Śyāvavarṇa, Kaṇḍu, Bahusrava*	Weeping Eczema	Dry crusts Tiny vesicles Itchy and inflamed skin Weep & ooze golden yellow or clear fluid
8	*Pāmā*	*Doṣa- Pitta Kapha, Śveta Arūṇa, Śyāvapiḍakā Kaṇḍu*	Scabies	Tiny reddish Blisters Intense itchying
9	*Carmadala*	*Doṣa- Pitta Kapha Rakta Sphota, Kaṇḍu, Rūjā*	Tinea Pedis	Rash between toes Redness Itching with peeling, fissuring and maceration
10	*Dadru*	*Doṣa- Pitta Kapha Rāga Maṇḍala, pīḍikā Kaṇḍu*	Ringworm	Red raised Blisters Round ring shape Itching
11	*Alasaka*	*Doṣa - Vāta Kapha, Rāga gaṇḍa, Kaṇḍu*	Prurigo Nodularis (PN)	Red, Hard lumps, Intensely Pruritus causing people to scratch themselves to point of bleeding or pain.

MCQs

1. *Dosha* predominance of *Dadru*
   a. *Kapha-Pitta* b. *Vata-Pitta*
   c. *Vata-Kapha* d. *Kaphaja*
2. *Dosha* predominance of *Sidhma*
   a. *Kapha-Pitta* b. *Vata-Pitta*
   c. *Vata-Kapha* d. *Kaphaja*
3. Resemblance with *Gunja* is seen in
   a. *Dadru* b. *Pundarika*
   c. *Charmadala* d. *Kakananthika*
4. Colour of *Kakanaka* is
   a. *Rakta-Krishna* b. *Krushna-Aruna*
   c. *Krushna* d. *Aruna*
5. *Aruna Kushtha* according to *Susrutha* is
   a. *Kshudra Kushtha* b. *Kaphaja Kushtha*
   c. *Mahakushtha* d. *Pittaja kushtha*
6. *Parisarpa* according to *Susrutha* is
   a. *Vatika* b. *Paittika*
   c. *Kaphaja* d. *Vata-Kaphaja*
7. *Vicarcika* according to *Susrutha* is
   a. *Vataja* b. *Pittaja*
   c. *Kaphaja* d. *Vata-Kaphaja*
8. *Rishyajihva Kushta* is
   a. *Vataja* b.*VataKaphaja*
   c. *Vatapittaja* d. *Pittaja*
9. *Varna* of *Eka-Kushta* is
   a. *Snigdha-Krushna* b. *Krushna-Aruna*
   c. *Sita-Aruna* d. *Atasipushpa-Varna*
10. *Varna* of *Kitibha* is
   a. *Snigdha-Krushna* b. *Krushna-Aruna*
   c. *Sita-Aruna* d. *Atasipushpa-Varna*

11. *Varna* of *Pundarika* is

a. *Snigdha-Krushna* b.*Krushna-Aruna*

c. *Sashwetam raktaparyantam*

d. Atasipushpa-*Varna*

12. *Varna* of *Dadru* is

a. *Snigdha-Krushna* b. *Krushna-Aruna*

c. *Sita-Aruna* d. *Atasipushpa-Varna*

13. *Kakananthika* has resemblance with

a. *Gunja Phala* b. *Atasipushpa*

c. *Matsya* d. *Gaja-Carma*

14. विस्तृतासमपर्यन्तं हृषितैर्लोमभिश्चितम् । तोदढ्यमल्पकण्डूकं are the lakshana of

a. *Dadru* b. *Kapala Kushta*

c. *Kakananthik* d. *Pundarika*

15. दीर्घप्रताना दूर्वावदतसीकुसुमच्छविः । उत्सन्नमण्डला are the lakshana of

a. *Dadru* b. *Kapala Kushta*

c. *Kakananthik* d. *Pundarika*

16. *Angulipatana karna nasa* occurs in

a. *Vatottara Kushta* b. *Pittottara Kushta*

c. *Kaphottara Kushta*

d. *Vata kaphaja Kushtha*

17. *Jantutpatti krimijanma* occurs in

a. *VatottaraKushta* b. *Pittottara Kushta*

b. *KaphottaraKushta* d.Vata kaphaja Kushtha

18. *Dosha* predominance of *Rakasa* according to *Nyayachandrika* is

a. *Vata* b. *Kapha*

c. *Pitta* d. *Vata-Kapha*

19. कौण्यं गतिक्षयोऽङ्गानां दलनं स्याच्च is a feature of which *dhatugatakushta* as per *Ashtanga Hridaya*

a. *Shukra* b. *Rakta*

c. *Mamsa* d. *Medas*

20. *Dosha* predominance of *Kacchu* according to *Nyayachandrika* is

a. *Vata* b. *Kapha*

c. *Pitta* d. *Vata-Kapha*

21. Consumption of Cilicima fish with milk causes

a. *Kushta* b. *Prameha*

c. *Rakta-Pitta* d. *Jwara*

Answer

1.	a	2.	c	3.	d
4.	a	5.	c	6.	a
7.	b	8.	c	9.	b
10.	a	11.	c	12.	d
13.	a	14.	b	15.	a
16.	b	17.	b	18.	b
19.	d	20.	c	21.	a

CHAPTER 30

Sheetapitta

Introduction

Urticaria. Allergic rashes. A skin disease. The appearance of intensity itching wheels over the body, having elevated centers surrounded by the patches of redness is identified as *Śītapitta*. The wheels tend to disappear in a day or two and sometimes accompanied by fever, vomiting and burning sensation. *Śītapitta* is caused by contact with very cold breeze, *kapha* and *vāta* getting increased, along with *pitta* also begin to travel externally in the skin and internally in blood and other tissue. Prodromal features of the disease include feeling of thirst, loss appetite, oppression in the chest, debility, heaviness of the body and redness of the eyes are its symptoms.

शीतमारुतसंस्पर्शात्प्रदुष्टौ कफमारुतौ ।
पित्तेन सह सम्भूय बहिरन्तर्विसर्पतः ॥
M.Ni. 50/1

Kapha and *Vāta* become aggravated due to exposure to cold breeze circulating all over the body (i.e. both externally & internally) in association with *pitta* causes *Śitapitta.*

शीतपित्त पूर्वरूप (*Śīta-Pitta Pūrvarupa)*

पिपासाऽरुचिहृल्लासदेहसादाङ्गगौरवम् । रक्तलोचनता
तेषां पूर्वरूपस्य लक्षणम् ॥
M.Ni. 50/2

Premonitory signs of symptoms of *Śitapitta* are thirst, anorexia, oppression in the chest, debility, heaviness & reddishness of eyes.

शीतपित्त लक्षण (*Udarda/Śīta-Pitta Lakṣana)*

वरटीदष्टसंस्थानः शोथः संजायते बहिः ।
सकण्डूस्तोदबहुलश्छर्दिज्वरविदाहवान् ॥
उदर्दमिति तं विद्याच्छीतपित्तमथापरे ।
वाताधिकं शीतपित्तमुदर्दस्तु कफाधिकः ॥
M.Ni. 50/3-4

Skin eruption which are elevated resemble like that produced by the sting of the wasp, associated with severe itching, pricking pain, vomiting, fever, burning sensation are the characteristic features of *Udarda* & some people says it as the *Śītapitta*. Prominent distinguishing feature between *Udarda* and *Śītapitta* is predominance of *Doṣas* i.e. *Vāta* is predominant in *Śītapitta* while in *Udarda Kapha* is predominant.

उदर्द (Udarda)

सोत्सङ्गैश्च सरागैश्च कण्डूमद्भिश्च मण्डलैः ।
शैशिरः कफजो व्याधिरुदर्द इति कीर्तितः ॥
M.Ni. 50/5

Udarda is characterised by elevated, reddish, highly irritating, round patches produced by Kapha during *Śiśira ṛtu*.

कोठ (Koṭha)

असम्यग्वमनोदीर्णपित्तश्लेष्मान्ननिग्रहैः ।
मण्डलानि सकण्डूनि रागवन्ति बहूनि च ।
उत्कोठः सानुबन्धश्च कोठ इत्यभिधीयते ॥
M.Ni. 50/6

Koṭha manifest due to improper administration of emesis, suppresion of the movement of *Pitta, Kapha,* & *Anna* causes the development of innumerable rashes which are red associated with itching & relapse many times. This is known as *Koṭha.*

Differential diagnosis of Śītapitta, Udarda and Koṭha

S.No.	*Śītapitta*	*Udarda*	*Koṭha*
Doṣa Etiology	*Vāta* predominance exposure to cold breeze *Kapha* and *Vāta* become aggravated due to exposure to cold breeze circulating all over the body (i.e. both externally & internally) in association with *pitta* causes *Śītapitta*	*Kapha* predominance Excess *Kapha* during *Śiṣira ṛtu*	*Pitta, Kapha* nigraha *Koṭa* manifest due to improper administration of emesis, suppression of the movement of *Pitta, Kapha,* & *Anna*
Clinical presentation	Skin eruption which are elevated resemble like that produced by the sting of the wasp, associated with severe itching, pricking pain, vomiting, fever, burning sensation	*Udarda* is characterized by elevated, reddish, highly irritating, round patches produced by *Kapha* during *Śiṣira ṛtu.*	Developments of innumerable rashes which are red associated with itching & relapse many times. This is known as *Koṭha*
Contemporary	Hives /Urticaria	Hives /Urticaria	Rashes

MCQs

1. *Raktalochana* is seen in
   a. *Sitapitta Rupa*
   b. *Sitapitta Purvarupa*
   c. *Udarda*
   d. *Kotha*
2. *Dosha* predominance of *Udarda*
   a. *Vatapradhanatridosha*
   b. *Pitta pradhanatridosha*
   c. *Kaphapradhanatridosha*
   d. *Samasannipatika*
3. *Varadidashtasamsthana* is the feature of
   a. *Sitapitta*
   b. *Udarda*
   c. *Kotha*
   d. *Utkotha*

Answer

1.	b	2.	c	3.	a

CHAPTER
31

Shwitra

दारुणं चारुणं श्वित्रं किलासं नामभिस्त्रिभिः ।
विज्ञेयं त्रिविधं तच्च त्रिदोषं प्रायशश्च तत् ॥
दोषे रक्ताश्रिते रक्तं ताम्रं मांससमाश्रिते ।
श्वेतं मेदःश्रिते श्वेतं गुरु तच्चोत्तेरोत्तरम् ॥
यत् परस्परतोऽभिन्नं बहु यद्रक्तलोमवत् ।
यच्च वर्षगणोत्पन्नं तच्छ्वित्रं नैव सिध्यति ॥
अरक्तलोम तनु यत् पाण्डू नातिचिरोत्थितम् ।
मध्यावकाशे चोच्छूनं श्वित्रं तत्साध्यमुच्यते ॥
C.S.Ci. 7/173-176

Śvitra is caused by vitiation of all the three *Doṣās* but sometimes it may manifest by either single *Doṣa* or two *Doṣa.*

It is classified into 3 subtypes namely *Dāruṇa, Cāruṇa* and *Kilāsa.* Depending on the location it manifest symptoms i.e. patches are reddish, coppery and white in colour, if the morbidity is located in *Rakta, Māmsa* and *Meda* respectively. Subsequent are more severe.

साध्यासाध्यता *(Sādhyāsādhyatā)*

If the patches are mutually matted together having multiple lesions surrounded by red hairs with chronic duration are said to be incurable.

If the patches are surrounded by non reddish hairs having thin and white lesions with recent onset along with elevated margins between two patches are said to be curable. C.S.Ci. 7.175-176.

MCQs

1. Features of *Sadhya (Curable) Svitra*
   a. *Krishnaroma, Asamsrishtam & Atibahala*
   b. *Arunaroma, Asamsrishtam & bahala*
   c. *Pitaroma, samsrishtam & Abahala*
   d. *Asuklaroma, Asamsrishtam & Abahala*
2. *Roma vidwamsi* is the feature of which type of *Svitra*
   a. *Vatika* b. *Paittika*
   c. *Kaphaja* d. *Tridoshaja*
3. *Kilasa* is the synonym of
   a. *Svitra* b. *Kusta*
   c. *Vatarakta* d. *Arsas*

Answer

1.	d	2.	b	3.	a

CHAPTER
32

Visarpa

Introduction

A skin disease. Acute spreading suppurations. *Visarpa* is an indisposition characterised by a rapidly spreading inflammation of the skin; The main signs and symptoms of *visarpa* include rapid spreadings of inflammation of skin with a throbbing or prickly or stabbing pain, lassitude, horripilation, appearance of deep pink-coloured and waxy patches on the skin with itching. Wax (probably as removing that disease).

भगवन्! दारुणं रोगमाशीविषविषोपमम् ।
विसर्पन्तं शरीरेषु देहिनामुपलक्षये ॥
सहसैव नरास्तेन परीताः शीघ्रकारिणा ।
विनश्यन्त्यनुपक्रान्तास्तत्र नः संशयो महान् ॥
C.S.Ci. 21/6-7

Visapra is serious disorder afflicting like cobra venon spreading various parts of the body very quickly. Negligence or delay in treatment succumbs to death.

विविधं सर्पति यतो विसर्पस्तेन स स्मृतः ।
परिसर्पोऽथवा नाम्ना सर्वतः परिसर्पणात् ॥
C.S.Ci. 21/11

Lesion spreads either upwards or downwards or sidewards or in all the directions is the characteristic feature of *Visarpa*. It is called *Parisarpa* because it spreads all over the body.

विविधं सर्पति यतो विसर्पस्तेन स स्मृतः ।
परिसर्पोऽथवा नाम्ना सर्वतः परिसर्पणात् ॥
C.S.Ci. 21/11

कुर्वन्ति विस्तृतमनुन्नतमाशु शोफं तं सर्वतो विसरणाच्च विसर्पमाहुः ॥*S.S.Ni. 10/3*

Visarpa is characterised by quick manifestation of swelling, having their own special features, spreading to all parts of body, lesion is wide and doesn't develop swelling. It is called *Visarpa*, because it spreads to all the parts of the body.

विसर्प हेतु (*Visarpa Hetu*)

लवणाम्लकटूष्णानां रसानामतिसेवनात् ।
दध्यम्लमस्तुशुक्तानां सुरासौवीरकस्य च ॥
व्यामन्नबहुमद्योष्णरागषाडवसेवनात् ।
शाकानां हरितानां च सेवनाच्च विदाहिनाम् ॥
कूर्चिकानां किलाटानां सेवनान्मन्दकस्य च ।
दध्नः शाण्डाकिपूर्वाणामासुतानां च सेवनात् ॥
तिलमाषकुलत्थानां तैलानां पैष्टिकस्य च ।
ग्राम्यानूपौदकानां च मांसानां लशुनस्य च ॥
प्रक्लिन्नानामसात्म्यानां विरुद्धानां च सेवनात् ।
अत्यादानाद्दिवास्वप्नादजीर्णाध्यशनात् क्षतात् ॥
क्षतबन्धप्रपतनाद्धर्मकर्मातिसेवनात् ।
विषवातारग्निदोषाच्च विसर्पाणां समुद्भवः ॥
C.S.Ci. 21/16-21

- Excessive consumption of salt, sour, pungent and hot substances.
- Increased intake of sour curd, curdwater, vinegar, alcoholic beverages, sour drink, fermented wine (alcohol); condiments & pickles having hot substances.
- Regular intake of excess greeny vegetables, salads, soup preparations made up of hot substances, inspissated milk, cheese, immature curd, fermented preparations like *Śaṇḍāki* (a type of sour gruel), Sesamum, Black gram, horse gram, oily preparations (junk foods) and flour preparations (pastries).
- Consumption of meat of animals which are residing in domestic, marshy and aquatic regions. Regular intake of garlic, decomposed food, unsuitable and mutually contradictory food substances.
- Excess intake of food, eating during indigestion and frequently eating habits
- Indulging in day sleep, suffering from traumatic injuries. Falling of bandages from wounds. Excessive exposure to hot sun, physical exertion, poisonous air, strong breeze and fire.

देहे शीघ्रं विसर्पन्ति तेऽन्तरन्तास्थिता बहिः ।
बहिःस्था द्वितये द्विस्थाःविद्यात्तत्रान्तराश्रयम् ॥
मर्मोपतापात्सम्मोहादयनानां विघट्टनात् ।
तृष्णातियोगाद्वेगानां विषमं च प्रवर्तनात् ॥
A.H.Ni. 13/45-46

Etiological factors for bāhya (external) and Abhyantra (Internal) Visarpa : *Vāgbhaṭa* mentioned two kinds of etiology i:e one for internal variety second one for external variety. Internal variety of *Visarpa* manifest due to afflictions of vital organs, unconsciousness, trauma to the sensory organs, excessive thirst, improper initiation of urges of body, sudden loss of strength & digestive fire. External variety of *Visarpa* is manifest due to the factors which are opposite to the causation of internal variety of *Visarpa.*

विसर्प संप्राप्ति (*Samprāpti of Visarpa*)

एतैर्निदानैव्यामिश्रैः कुपिता मारुतादयः ।
दूष्यान् संदूष्य रक्तादीन् विसर्पन्त्यहिताशिनाम् ॥
C.S.Ci. 21/22

त्वङ्मांसशोणितगताः कुपितास्तु दोषाः सर्वाङ्गसारिणमिहास्थित-मात्मलिङ्गम् । कुर्वन्ति विस्तृतमनुन्नतमाशु शोफं तं सर्वतो विसरणाच्च विसर्पमाहुः ॥ *S.S.Ni. 10/3*

One who indulges in unwholesome diet along with other etiological factors mentioned above leading to aggravation of all the *Vātādi* three *Doṣās,* which inturn vitiates *Rakta, Lasikā, Tvak* and *Māmsa* and manifest *Visarpa*.

It is charcterised by quick manifestation of non bulging skin eruptions having their own special features, spreads to all parts of body with wider lesions.

तत्र दोषा यथायथम् । प्रकोपणैः प्रकुपिता विशेषेण विदाहिभिः ॥ देहे शीघ्रं विसर्पन्ति तेऽन्तरन्तास्थिता बहिः । बहिःस्था द्वितये द्विस्थाः विद्यात्तत्रान्तराश्रयम् ॥ *A.H.Ni 13/44-45*

Excacerbation of *Doṣās* by their respective causative factors especially *Pitta* aggravating factors like *Vidāhi* etc. leads to quick spreading of *Doṣās* to internal path and external path.

Doṣa-dūṣya Sammūrchanā takes place either in *Bāhya* or *Abhyantara* path leading to development of respective *Bāhya* & *Abhyantara Visarpa.*

रक्तं लसीका त्वङ्मांसं दूष्यं दोषास्त्रयो मलाः ।
विसर्पाणां समुत्पत्तौ विज्ञेयाः सप्त धातवः ॥
C.Ci. 21/15

Seven components namely *Vāta, Pitta, Kapha, Rakta, Lasikā, Māmsa* and *Tvak* are mainly responsible for the manifestation of *Visarpa.*

Nidān Sevana (Consumption of
Etiological factors)
↓
Aggravation of *Pitta, Śleṣma* and *Vāta*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro
intestinal tract
↓
Excacerbation of *Doṣās* by their respective
causative factors especially *Pitta* aggravating
factors like *Vidāhi* etc. leads to quick spreading
of *Doṣās* to internal path and external path.
↓
Vitiates *Rakta, Lasikā, Māmsa* and *Tvak*
↓
Doṣa-dūṣya Sammūrchanā takes place either in
Bāhya or *Abhyantara* path leading to
↓
Bāhya Visarpa. or *Abhyantara Visarpa.*

(It is charcterised by quick manifestation of non bulging skin eruptions having their own special features, spreads to all parts of body with wider lesions)

विसर्प संप्राप्ति घटक (*Samprāpti Ghaṭaka of Visarpa*)

Doṣa : तत्र विसर्पस्य पित्तप्रबलं सप्र द्रव्यं ।
S.S.Ni. 5/3 (Nyāya Candrika)
Pitta Pradhāna Tridoṣaja
Vāta→ *Vyāna*
Pitta→ *Pācaka, Bhrājaka*
Kapha→ *Kledaka*

Dūṣya : *Dhātu*→ Initially : *Tvak (Rasa), Rakta, Māmsa* and *Lasikā*
Upadhātu→ *Sirā, Snāyu, Tvak*

Śārīra Mala → Mūtra & Purīṣa
Dhātu Mala → Sveda, Nakha.

Agni : *Jaṭharāgni, Dhātvāgni*
Agnidușți : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Rasavaha, Raktavaha, Māṁsavaha,*
Srotoduṣṭi : *Saṅga*
Adhiṣṭhāna : *Abhyantara* and *Bāhya Roga Mārga*
Sancārasthāna : *Tvak* & other *Dhātus*
Vyaktasthāna : *Tvacā*
Udbhavasthāna : *Āmāśayottha, Śākhā-Tvak*
Svabhāva : *Āśukāri, Cirakāri*
Prabhāva : *Eka Doṣaja* Curable *Agni Visarpa, Kardama Visarpa, Granthi Visarpa, Sannipātaja Visarpa* are incurable.
Roga Bheda : 7 Varieties
Roga Mārga : *Abhyanta, Bāhya* & *Madhyama*

अधिष्ठान (*Adhiṣṭhāna*)

बहि:श्रित: श्रितश्चान्तस्तथा चोभयसंश्रित: ।
विसर्पो बलमेतेषां ज्ञेयं गुरु यथोत्तरम् ॥
C.S.Ci. 21/23

Place of manifestation of *Visarpa* is external, internal and both. It is difficult to cure in their progressive order.

अन्त:स्थित विसर्प लक्षण (*Antaḥsthita Visarpa Lakṣaṇa*)

मर्मोपघातात् संमोहादयनानां विघट्टनात् ।
तृष्णातियोगाद्वेगानां विषमाणां प्रवर्तनात् ॥
विद्याद्विसर्पमन्तर्जमाशु चग्निबलक्षयात् ।
अतो विपर्ययाद्बाह्यमन्यैर्विद्यात् स्वलक्षणैः ॥
C.S.Ci. 21/26-27

Internal variety of *Visarpa* is characterised by afflictions of *Marma* (vital organs), unconsciousness, obstruction in channels, severe thirst, irregularity in the release of urges and sudden diminution of internal digestive fire.

विसर्प भेद (*Visarpa Bheda*)

स च सप्तविधो दोषैर्विज्ञेय: सप्तधातुक: ।
पृथक् त्रयस्त्रिभिश्चैको विसर्पो द्वन्द्वजास्त्रय: ॥
वातिक: पैत्तिकश्चैव कफज: सान्निपातिक: ।
चत्वार एते वीसर्पा वक्ष्यन्ते द्वन्द्वजास्त्रय: ॥
आग्नेयो वातपित्ताभ्यां ग्रन्थ्याख्य: कफवातज: ।
यस्तु कर्दमको घोर:स पित्तकफसंभव: ॥
C.S.Ci. 21/12-14

Visarpa is classified into seven subtypes namely 1. *Vātaja*, 2. *Pittaja*, 3. *Kaphaja*, 4. *Sannipātika*, 5. *Āgneya (Vāta-Pittaja)*, 6. *Granthi (Kapha-Vātaja)* and 7. *Kardamaka (Pitta-Kaphaja)*.

वातज विसर्प हेतु एवं लक्षण (*Vātaja Visarpa Hetu evam Lakṣaṇa*)

रूक्षोष्णै: केवलो वायु: पूरणैर्वा समावृत: ।
प्रदुष्टो दूषयन् दूष्यान् विसर्पति यथाबलम् ॥

तस्य रूपाणिभ्रमदवथुपिपासानिस्तोदशूलाङ्गमर्दोद्वेष्टनकम्प- ज्वरतमककासास्थिसंधिभेदविश्लेषणवेपनारोचकाविपाकाश्चक्षुषोराकुलत्वमस्त्रागमनं पिपीलिकासंचार इव चाङ्गेषु यस्मिंश्चावकाशे विसर्पो विसर्पति सोऽवकाश: श्यावारुणाभास: श्वयथुमान् निस्तोदभेदशूलायामसंकोचहर्षस्फुरणैरतिमात्रं प्रपीड्यते, अनुपक्रान्तश्चोपचीयते शीघ्रभेदै: स्फोटकैस्त- नुभिररुणाभै: श्यावैर्वा तनुविशदारुणाल्पास्त्रावै:, विबद्धवातमूत्रपुरीषश्च भवति, निदानोक्तानि चास्य नोपशेरते विपरीतानि चोपशेरत इति वातविसर्प: ॥
C.S.Ci. 21/29-30

Vātaja Visarpa manifest by following etiological factors.

Excess consumption of ununctous and hot substances and occlusion by vitiated *Pitta-Kapha* due to over nourishment and vitiates *Dūṣyās* and manifest *Vātaja Visarpa* and it spreads to entire body depending on the nature of severity. Hot substances are specially mentioned for the manifestation of *Vātaja Visarpa* along with ununctous substances, it indicates that there is role of *Pitta* even in *Vātaja* type of *Visarpa*.

Symptomatology

It is charcterised by giddiness, burning sensation in eyes, thirst, pricking pain, colic pain, malaise, cramps, tremors, fever, feeling of darkness in front of eyes, cough, breaking pain in bones and joints and their dislocation, shivering, anorexia, indigestion, congestion of eyes, lachrymation, and there is a feeling as if ants are crawling over the body.

The part where the disease spreads becomes greyish, blackish, reddish. Patient suffer from intense pain which is either pricking, breaking, colic, expansion, contraction, tingling sensation (horripilation) and throbbing sensation.

Due to negligency or delay in treatment produces various kinds of afflictions i:e quick bursting, cracking, erupted lesions are small in size, having blackish or reddish discolouration with thin, transparent pink and scanty discharge.

Patients suffers from retention of flatus, urine and stool. Symptoms aggravate after exposure to etiological factors and alleviates after adoption of etiology opposite to causative ones.

पित्तज विसर्प हेतु एवं लक्षण (*Pittaja Visarpa Hetu evam Lakṣaṇa*)

पित्तमुष्णोपचारेण विदाह्यम्लाशनैश्चितम् । दूष्यान् संदूष्य धमनीः पूरयन् वै विसर्पति ॥

तस्य रूपाणिज्वरस्तृष्णा मूर्च्छा मोहश्छर्दिररोचकोऽङ्गभेदः स्वेदोऽतिमात्रमन्तर्दाहः प्रलापः शिरोरुक् चक्षुषोराकुलत्वमस्वप्नमरतिर्भ्रमः शीतवातवारितर्षोऽतिमात्रं हरितहारिद्रनेत्रमूत्रवर्चस्त्वं हरित हारिद्ररूपदर्शनं च, यस्मिंश्चावकाशे विसर्पोऽनुसर्पति सोऽवकाशस्ताम्रहरितहारिद्रनील- कृष्णरक्तानां वर्णानामन्यतमं पुष्यति, सोत्सेधैश्चातिमात्रं दाहसंभेदनपरीतैः स्फोटकैरुपचीयते तुल्यवर्णास्रावैरचिरपाकैश्च, निदानोक्तानि चास्य नोपशेरते विपरीतानि चोपशेरत इति पित्तविसर्पः ॥

C.S.Ci. 21/31-32

Etiology : *Pittaja Visarpa* manifest due to consumption of hot substances, like *Vidāhi* and *Āmla* etc. leading to excacerbation of *Pitta,* which in turn vitiates *Dhātus,* vitiated *dhātus* spreads the disease to entire body.

Symptoms : It is characterised by fever, thirst, fainting, unconsciousness, vomiting, anorexia, breaking pain in limbs, excessive sweating and burning sensation inside the body, delirium, headache, congestion in the eyes, sleeplessness, dislike for everything or restlessness, giddiness, intense desire for cold wind and water.

Eyes, urine and stool becomes deep green and yellowish discolouration and objects appear either green or yellow.

Affected area where the disease located develops coppery, green, yellow, blue, black and red coloured raised skin eruptions having intense burning sensation and breaking pain, suppurating quickly and discharges exudates of similar colour like that of eruptions. Symptoms excacerbates after consumption of factors which aggravates *Pitta* and alleviation of symptoms takes place after consumption of factors which are opposite to the cause of the disease.

कफज विसर्प हेतु एवं लक्षण (*Kaphaja Visarpa Hetu evam Lakṣaṇa*)

स्वाद्वम्ललवणस्निग्धगुर्वन्नस्वप्नसंचितः ।
कफः संदूषयन् दूष्यान् कृच्छ्रमङ्गे विसर्पति ॥

C.Ci. 21/33

तस्य रुपाणि शीतकः शीतज्वरो गौरवं निद्रा तन्द्राऽरोचको मधुरास्यत्वमास्योपलेपो निष्ठीविका छर्दिरालस्यं स्तैमित्यमग्निनाशो दौर्बल्यं च, यस्मिश्चावकाशे विसर्पोऽनुसर्पति सोऽवकाशः श्वयथुमान् पाण्डुर्नातिरक्तः स्नेहसुप्तिस्तम्भगौरवैरन्वितोऽल्पवेदनः कृच्छ्रपाकैश्चिरकारिभिर्बहुलत्वगुपलेपैः स्फोटैः श्वेतपाण्डुभिरनुबध्यते, प्रभिन्नस्तु श्वेतं पिच्छिलं तन्तुमद्घनमनुबद्धं स्निग्धमास्रावं स्रवति, ऊर्ध्वं च गुरुभिः स्थिरैर्जालावततैः स्निग्धैर्बहुलत्वगुपलेपैर्व्रणैरनुबध्यतेऽनुषङ्गी च भवति, श्वेतनखनयनवदनत्वङ्मूत्रवर्चस्त्वं निदानोक्तानि चास्य नोपशेरते विपरीतानि चोपशेरत इति श्लेष्मविसर्पः ॥ *(C.S.Ci. 21/34)*

Etiology for *Kaphaja Visarpa* : *Kaphaja* excacerbates due to consumption of ingredients which are sweet, sour, salt, unctuous, heavy and sleeping during day time and such other causative factors which aggravates *Kapha,* which in turn vitiates *Dūṣyās* and manifest *Kaphaja Visarpa,* which spreads slowly.

Symptoms : It is characterised by feeling of cold, fever with chills, heaviness, excessive sleep, drowsiness, anorexia, sweet taste in mouth, sweetishness and adherence of sticky material in oral cavity, splitting of saliva, vomiting, lassitude, timidity, diminution of digestive fire and strength.

The part in which disease spreads becomes oedematous, pale-yellow, slightly reddish and unctuous associated with numbness, stiffness, heaviness and mild pain.

Suppuration of the skin eruption is slow, chronic and they appear in large number; the skin over these pustules gets covered with white or pale yellow coloured sticky material.

When pustules burst they discharge white, slimy, thready, viscous, knotty and unctuous materials. As the disease advances the area is covered with wounds which are heavy (deep seated), stable, surrounded by capillary network, unctuous and covered with copious smear over skin appear and re-appear. Wounds remain for longer period of time.

Whitishness of nails, eyes, face, skin, urine, and stool.

The etiological factors are non conducive to *Kaphaja Visarpa* and opposite to etiology are conducive to *Kaphaja Visarpa* patient.

अग्नि विसर्प हेतु एवं लक्षण (*Agni Visarpa Hetu evam Lakṣaṇa)*

वातपित्तं प्रकुपितमतिमात्रं स्वहेतुभिः।
परस्परं लब्धबलं दहद्गात्रं विसर्पति ॥

तदुपतापादातुरः सर्वशरीरमङ्गारैरिवाकीर्यमाणं मन्यते, छर्द्यतीसारमूर्च्छादाहमोहज्वर तमकारोचकास्थिसंधिभेदतृष्णाविपाकाङ्गभेदादिभिश्चाभिभूयते, यं यं चावकाशं विसर्पोऽनुसर्पति सोऽवकाशः शान्ताङ्गारप्रकाशोऽतिरक्तो वा भवति, अग्निदग्धप्रकारैश्च स्फोटैरुपचीयते, स शीघ्रगत्वादाश्वेवमर्मानुसारी भवति, मर्मणि चोपतप्ते पवनोऽतिबलो भिनत्त्यङ्गान्यतिमात्रं प्रमोहयति संज्ञां, हिक्काश्वासौ जनयति नाशयति निद्रां, स नष्टनिद्रः प्रमूढसंज्ञो व्यथितचेता न क्वचन सुखमुपलभते, अरतिपरीत स्थानादासनाच्छय्यां क्रान्तुमिच्छति, क्लिष्ठभूयिष्ठश्चाशु निद्रां भजति, दुर्बलो दुःखप्रबोधश्च भवति तमेवंविधमग्निविसर्पपरीतमचिकित्स्यं विद्यात् ॥

C.S.Ci. 21/35-36

Etiology : Simultaneous aggravation of *Vāta* and *Pitta* by their respective causative factors, gain strength from each other and spread all over the body and produces severe burning sensation is called *Visarpa.*

Symptoms : It is charcterised by burning sensation all over the body and patient feels as if his body is sprinkled with flaming charcoals associated with vomiting, diarrhoea, fainting, burning sensation, unconsciousness, fever, feeling as if entering into darkness, anorexia, pain in the bones and joints, morbid thirst, indigestion, breaking pain all over the body etc.

The part where disease manifested appears like extinguished charcoal or excessive redness and it is surrounded by skin eruptions, which resemble like those caused by burns. Due to acute spreading nature it afflicts vital organs badly as a result *Vāta* gets aggravated excessively and produce breaking pain in the limbs, unconsciousness, hiccup, breathlessness, insomnia, mental confusion due to sleeplessness leads miserable life and doesn't find comfort anywhere. Due to discomfort in any of the positions, he changes his place & seat oftenly and always desires to go to bed. Being afflicted with miseries, he gets sleep quickly and the weak one is aroused with difficulty. It is incurable.

कर्दम विसर्प हेतु एवं लक्षण (**Kardama Visarpa Hetu evam Lakṣaṇa**)

कफपित्तं प्रकुपित्तं बलवत् स्वेन हेतुना।
विसर्पत्येकदेशे तु प्रक्लेदयति देहिनम् ॥

तद्विकाराः शीतज्वरः शिरोगुरुत्वं दाहः स्तैमित्यमङ्गावसदनं निद्रा तन्द्रा मोहोऽन्नद्वेषः प्रलापोऽग्निनाशो दौर्बल्यमस्थिभेदो मूर्च्छा पिपासा स्रोतसां प्रलेपो जाड्यमिन्द्रियाणां प्रायोपवेशनमङ्गविक्षेपोडङ्गमर्दोऽरतिरौत्सुक्यं चोपजायते, प्रायश्चामाशये विसर्पत्यलसक एकदेशग्राही च , यस्मिंश्चावकाशे विसर्पो विसर्पति सोऽवकाशो रक्तपीतपाण्डुपिडकावकीर्ण इव मेचकाभः कालो मलिनः स्निग्धो बहूष्मा गुरुः स्तिमितवेदनः श्वयथुमान् गम्भीरपाको निरास्रावः शीघ्रक्लेदः स्विन्नक्लिन्नपूतिमांसत्वक् क्रमेणाल्परुक् परामृष्टोऽवदीर्यते कर्दम इवावपीडितोऽन्तरं प्रयच्छत्युपक्लिन्नपूतिमांसत्यागी सिरास्नायुसंदर्शी कुणपगन्धी च भवति संज्ञास्मृतिहन्ता च; तं कर्दमविसर्पपरीतमचिकित्स्यं विद्यात् ॥ *C.S.Ci. 21/37-38*

Etiology : Simultaneous aggravation of *Kapha & Pitta* by their respective etiological factors, spread to localised parts and brings moistening in the affected parts.

Symptoms : It is characterised by cold, fever, heaviness in head, burning sensation, timidity, debility in body parts, sleep, drowsiness, unconsciousness, dislike for food, delirium, diminution of loss of digestive fire, debility, breaking pain in bones, fainting, thirst, adhesion of sticky material inside the *Srotas,* inability to perform normal functions by sensory organs, constant feeling for voiding stool, stretching of extremities, general malaise, restlessness and anxiety. It spreads to *Āmāśaya* in a slow manner and gets localised there.

The part where disease manifested appears like either red, yellow and pale yellow colour associated with muddy, black, dirty and unctuous. It induces warmthness, heaviness, mild pain which is continuous, swelling and deep seated suppuration. Skin eruptions are free from exudations but manifest quick moistening, sweating, sloughing of muscles and skin; the pain over the affected lesion is less and it appears gradually; after rubbing skin eruption bursts, and when pressed, sticky and putrified muscle tissue comes out; after removing dirt and slough, blood vessels & ligaments become visible and cadaverous smell emits from the affected parts. Patient become unconscious and looses memory. It is incurable

ग्रन्थि विसर्प हेतु एवं लक्षण (Granthi Visarpa Hetu evam Lakṣaṇa)

स्थिरगुरुकठिनमधुरशीतस्निग्धान्नपानाभिष्यन्दिसेविनामव्यायामादिसेविनामप्रतिकर्मशीलानां श्लेष्मा वायुश्च प्रकोपमापद्यते, तावुभौ दुष्टप्रवृद्धावतिबलौ प्रदूष्य दूष्यान् विसर्पाय कल्पेते; तत्र वायुः श्लेष्मणा विबद्धमार्गस्तमेव श्लेष्माणमनेकधाभिन्दन् क्रमेण ग्रन्थिमालां कृच्छ्रपाकसाध्यां कफाशये संजनयति, उत्सन्नरक्तस्य वा प्रदूष्य रक्तं सिरास्नायुमांसत्वगाश्रितं ग्रन्थीनां मालां कुरुते तीव्ररुजानां स्थूलानामणूनां वा दीर्घवृत्तरक्तानां, तदुपतापाज्ज्वरातिसारकासहिक्काश्वासशोषप्रमोहवैवर्ण्यारोचकाविपाकप्रसेकच्छर्दिमूर्च्छाङ्गभङ्गनिद्रारतिसदनाद्याः प्रादुर्भवन्त्युपद्रवाः; स एतैरुपद्रुतः सर्वकर्मणां विषयमतिपतितो विवर्जनीयो भवतीति ग्रन्थिविसर्पः ॥

C.S.Ci. 21/39

Etiology : Simultaneous aggravation of *Kapha* & *Vāta* by their respective etiological factors, vitiate *Dūṣyās* and manifest *Granthi Viśarpa.*

Consumption of food and drinks which are solid, heavy, hard, sweet, cold, unctuous and channel blocking substances. Lack of physical activity and not undergoing biopurificatory procedures at regular interval.

Due to above etiological factors *Kapha* aggravates and accumulates in excess in the channel of circulation as a result *Vāta* gets obstruction for its normal movement leading to its aggravation. Aggravated *Vāta* disintegrates *Kapha* into several parts gradually giving rise to a series of *Granthis* in the seats of *Kapha.* These *Granthis* hardly suppurate and difficult for management.

Symptoms : If person adopts *Rakta* provocative measures along with *Vāta* & *Kapha* aggravating factors leading to vitiation of *Rakta* along with *Vāta* & *Kapha* giving rise to a series of glands in blood vessels, ligaments, muscles and skin. These glands are exceedingly painful, either big or small or elongated or round having reddish discolouration.

Patient also suffers from fever, diarrhoea, cough, hiccup, dyspnoea, consumption, unconsciousness, deranged complexion, anorexia, indigestion, salivation, vomiting, fainting, breaking of body parts, sleep, restlessness, malaise etc. Such patients transcends all the therapeutic procedures. That's why treatment to these kinds of patients must be rejected.

सन्निपातिक विसर्प (Sannipātika Visarpa)

सर्वायतनसमुत्थं सर्वलिङ्गव्यापिनं सर्वधात्वनुसारिणमाशुकारिणं महात्ययिकमिति सन्निपातविसर्पमचिकित्स्यं विद्यात् ॥

C.S.Ci. 21/41

Simultaneous aggravation of *Vāta, Pitta* and *Kapha* by their respective etiological factors leads to presentation of symptoms of all the *Doṣās* and spreads quickly to all body parts & manifest severe serious diastrous condition and becomes incurable.

क्षतज विसर्प (Kṣataja Visarpa)

सद्यः क्षतव्रणमुपेत्य नरस्य पित्तं रक्तञ्च दोषबहुलस्य करोति शोफम् ।श्यावं सलोहितमतिज्वरदाहपाकस्फोटैः कुलत्थसदृशैरसितैश्च कीर्णम् ॥ *S.S.Ni. 10/7*

बाह्यहेतोः क्षतात्क्रुद्धः सरक्तं पित्तमीरयन् ॥

विसर्पं मारुतः कुर्यात् कुलत्थसदृशैश्चितम् ।
स्फोटैः शोफज्वररुजादाहाढ्यं श्यावलोहितम् ॥
A.H. Ni. 13/65-66

Vāta aggravates by external causative factors like trauma leading to exacerbation of *Rakta* and *Pitta* produces *Kṣataja Visarpa*. It is characterized by swelling, which is either bluish or slightly reddish and surrounded by black eruptions resembling horsegram associated with bleeding, fever, burning sensation, and pus formation.

उपद्रव (Upadrava)

Complications of *Visarpa* are as follows:-

पृथग्दोषैस्त्रयः साध्या द्वन्द्वजाश्चानुपद्रवाः ।
असाध्यौ क्षतसर्वोत्थौ सर्वे चाक्रान्तमर्मकाः ॥
शीर्णस्नायुसिरामांसाः प्रक्लिन्नाः शवगन्धयः ।
A.H.Ni. 13/67-68

Involvement of all the three *Doṣās;* disease spreading to vital organs; loss of tendons, veins and muscles and these are highly moist and cadeveric smell emits from these areas.

साध्यासाध्यता (Sādhyāsādhyatā)

सिध्यन्ति वातकफपित्तकृता विसर्पाः सर्वात्मकः क्षतकृतश्च न सिद्धिमेति। तैलानिलावपि च दर्शितपूर्वलिङ्गौ सर्वे च मर्मसु भवन्ति हि कृच्छ्रसाध्याः ॥ *S.S.Ni. 10/8*

तत्र वातपित्तश्लेष्मनिमित्ता विसर्पास्त्रयः साध्या भवन्ति; अग्निकर्दमाख्यौ पुनरनुपसृष्टे मर्मणि अनुपगते वा सिरास्नायुमांसक्लेदे साधारणक्रियाभिरुभावेवाभ्यस्यमानौ प्रशान्तिमापद्येयाताम्, अनादरोपक्रान्तः पुनस्तयोरन्यतरो हन्याद्देहमाश्वेवाशीविषवत्; तथा गन्थिविसर्पमजातोपद्रवमारभेत चिकित्सितुम् उपद्रवोपद्रुतं त्वेनं परिहरेत्; सन्निपातजं तु सर्वधात्वनुसारित्वादाशुकारित्वाद्विरुद्धोपक्रमत्वाच्चासाध्यं विद्यात् ॥ *C.S.Ci. 21/42*

Visarpa manifested by single *Doṣa* either *Vāta* or *Pitta* or *Kapha* predominance is said to be curable. *Sannipātaja* and *Kṣataja Visarpa* are incurable. *Pitta-Vātaja Visarpa (Āgneya Visarpa)* with complete symptomatology indicates incurability of the disease.

All kinds of *Visarpa* become difficult to cure if vital organs are involved.

Agni Visarpa and *Kardama Visarpa* can be managed by the constant use of general therapeutic measures, provided vital organs are spared and there is no softening of the vessels, ligaments and muscle tissue.

Delayed or improper treatment employed to *Agni Visarpa* and *Kardama Visarpa* leads to death of a person like snake poison.

Granthi Visarpa without complications can be curable and with complications it is said to be incurable.

Sannipātaja Visarpa is incurable because it spreads to all vital organs and tissues and spreads quickly and line of treatment is antagonistic.

उपशय/पथ्य (Upaśaya/Pathya)

Vamana Karma, Mustādi Kvātha, Sārivādi Kvātha, Kiratatiktādi Kvātha, Rakta Mokṣaṇa.

Ghṛta : *Mahātiktaka Ghṛta, Trāyamāṇa Ghṛta, Gauryādi Ghṛta.*

Bāhya lepa : *Udumbarādi pradeha, Nyagrodādya lepa, Kālīyādi pralepa, Śādvalādi pradeha, Sārivādhya pralepa, Naladādya pralepa, Balādya lepa, Prapuṇḍarīkādya pralepa, Triphalādi pradeha, Khadirādyā lepana.*

Recipes for cleaning wounds

Paṭolādi kvātha, Paṭolādi ghṛta

पथ्य (Pathya)

अन्नपानानि वक्ष्यामि विसर्पाणां निवृत्तये ।
लङ्घितेभ्यो हितो मन्थो रुक्षः सक्षौद्रशर्करः ॥
मधुरः किंचिदम्लो वा दाडिमामलकान्वितः ।
सपरुषकमृद्वीकः सखर्जूरः शृताम्बुना ॥
तर्पणैर्यवशालीनां सस्नेहा चावलेहिका ।
जीर्णे पुराणशालीनां यूषैर्भुञ्जीत भोजनम् ॥
मुद्गान्मसूरांश्चणकान् यूषार्थमुपकल्पयेत् ।
अनम्लान् दाडिमाम्लान् वा पटोलामलकैः सह ॥
जाङ्गलानां च मांसानां रसांस्तस्योपकल्पयेत् ।
रुक्षान् परुषकद्राक्षादाडिमामलकान्वितान् ॥
रक्ताः श्वेता महाह्वाश्च शालयः षष्टिकैः सह ।
भोजनार्थे प्रशस्यन्ते पुराणाः सुपरिस्रुताः ॥
यवगोधूमशालीनां सात्म्यान्येव प्रदापयेत् ।

येषां नात्युचितः शालिर्नरा ये च कफाधिकाः ॥
C.S.Ci. 21/108-114

Food and drinks useful for curing *visarpa* are described below:

- Fasting is the beginning treatment for all varieties of *Visarpa.*
- Thereafter, he should be given *mantha* (roasted flour of cereals added with water in profuse quantity). It should be free from any unctuous material, but honey and sugar should be added. To make this drink sweet and sour, it should be added with *dāḍima, āmalaka, parūṣaka, mṛdvīkā* and *kharjūra*. The above mentioned flour of roasted cereals and other ingredients should be mixed with boiled water.
- The roasted flour of barley and *śāli* rice may be added with ghee, and it should be consumed in the form of linctus.
- After the above mentioned recipe is digested, the patient should be given boiled old rice along with vegetable soup. *Mudga, masūra* and *caṇaka* should be used for the preparation of this vegetable soup. To these pulses, *paṭola* and *āmalaka* may also be added for the preparation of the soup. This soup may not be made sour. But if the patient desires to take sour soup then add *dāḍima.*
- Red, white and *mahāhva* types of *śāli* rice and *ṣaṣṭika* type of rice, which are old and which are boiled with the residual water completely removed, should be given to the patient as food.
- Depending upon the eating habit (wholesomeness), the patient may be given either barley or wheat or *śāli* type of rice to eat. If *śāli* rice is not suitable and if *kapha* is aggravated in excess, then barley and wheat should be used as food. C.S.Ci 21.108-114.

Wholesome dietetics for patient of *Visarpa* are as follows:- *Laṅgana* (fasting), *Mantha* (churned drink) should be from unctuous material added with honey & sugar.

Mantha prepared with *Dāḍima, Āmalaka, Parūṣaka, Mṛdvikā* and *Kharjūra.* Patient should drink boiled water which is mixed with flour of roasted cereals and *Dāḍima, Āmalaka, Paruṣaka, Mṛdvikā* & *Kharjūra.*

Avalehika (paste like rice preparation) added with little fat (ghee) should be taken with *Mantha* prepared out of *Bārley* and *Śāli*. After the digestion of earlier recipe patient should take old *Śāli* rice with soups prepared out of *Mudga, Masūra, Caṇaka. Paṭola* and *Āmalaka* may also be added to the above soup but it should not be sour in taste. If patient desires sour taste soup, then the soup can be made by adding sour *Dāḍima.*

Meat soup prepared out of wild animals without fat and added with *Paruṣaka, Drākṣā, Dāḍima* and *Āmalaka.*

Red, white and *Mahāśāli* types of rice and *Ṣaṣṭika* type of rice, which are old. After removing residual water from boiled rice, then it should be given to patient for eating.

Considering one's own food habit the patient may be given either barely or wheat or *Śāli* type of rice for eating in case of *Kapha* predominant condition. *Śāli* type of rice is not wholesome, then barley & wheat should be used as food.

The following measures and materials are considered to be good, if taken in proper way and in right dosage for patients of *Visarpa*: *Virecana, Vamana, Lepa, Laṅgana* (fasting), *Rakta Mokṣana*, old barley, wheat, *Kanguni, Ṣaṣṭhi* and *Śāli* types of paddy, *Mudga, Masūra*, grams (*Caṇaka*), *Tura*, meat soup of wild animals and birds, butter, *Ghṛtam*, raisins, *Drākṣā, Kāravellaka, Vetāgra*, leaves of *Paṭola, Āmalakī, Khadira, Nāgakeśara, Lākṣa, Śirīṣa*, camphor, sandalwood, sesame seeds, *sugandhabālā, mustā* and pungent edibles. All the above are beneficial in curing *Visarpa* disorder. (B.R. 57/27-30)

The patients should not get excited and angry, they can be served with blood purifying materials, bitter eatables, the eatables that do not contain hot properties and the like liquids. All these materials should be used in treating a patient of *Visarpa.* (B.R. 57/31-32)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

विदाहीन्यन्नपानानि विरुद्धं स्वपनं दिवा ।
क्रोधव्यायामसूर्याग्निप्रवातांश्च विवर्जयेत् ॥

C.S.Ci. 21/115

The following food substances are unwholesome to *Visarpa* patients.

- Foods and drinks, which induces sour eructations, burning sensation, thirst and difficulty in digestion.
- Mutually antagonistic foods and drinks
- Sleeping during day time, anger, physical exercise. Excessive exposure to sun, fire & wind.

The following measures and edibles are strictly prohibited for the patients of *Visarpa*: exercising, sleeping during day time, intercourse, sitting or roaming in easterly winds or winds from any side, anger, grief, holding the urge to vomit, having jealous feeling on success of others, leafy vegetables, *Viruddhāśana*, curd, *Kurcika*, *Sauvīra* (*Kānji*), *Āsava-Ariṣṭa*, *Kilāṭa*, heavy to digest eatables and liquids, garlic, *Kulattha*, *Urada*, sesame, meat other than those of the wild animals, *Svedana* therapy, *Vidāhī Dravya*, salt, bitter and sour edibles, liquor and exposure to the bright sunlight. (B.R. 57/33-34).

Differential diagnosis of *Visarpa*

S.No.	*Vātaja*	*Pittaja*	*Kaphaja*	*Sannipātika*
Etiology	Excess consumption of ununctous and hot substances and occlusion by vitiated *Pitta-Kapha* due to over nourishment and vitiates *Dūṣyās* and manifest *Vātaja Visarpa* and it spreads to entire body depending on the nature of severity. Hot substances are specially mentioned for the manifestation of *Vātaja Visarpa* along with ununctous substances, it indicates that there is role of *Pitta* even in *Vātaja* type of *Visarpa*.	*Pittaja Visarpa* manifest due to consumption of hot substances, like *Vidāhi* and *Āmla* etc. leading to excacerbation of *Pitta*, which in turn vitiates *Dhātus*, vitiated *dhātus* spreads the disease to entire body.	*Kaphaja* excacerbates due to consumption of ingredients which are sweet, sour, salt, unctuous, heavy and sleeping during day time and such other causative factors which aggravates *Kapha*,	Simultaneous aggravation of *Vāta*, *Pitta* and *Kapha* by their respective etiological factors leads to presentation of symptoms of all the *Doṣās* and spreads quickly to all body parts & manifest severe serious diastrous condition and becomes incurable.
Clinical presentation	It is charcterised by giddiness, burning sensation in eyes, thirst, pricking pain, colic pain, malaise, cramps, tremors, fever, feeling of darkness in front of eyes, cough, breaking pain in bones and joints and their dislocation,	It is characterised by fever, thirst, fainting, unconsciousness, vomiting, anorexia, breaking pain in limbs, excessive sweating and burning sensation inside the body, delirium, headache, congestion in the	It is characterised by feeling of cold, fever with chills, heaviness, excessive sleep, drowsiness, anorexia, sweet taste in mouth, sweetishness and adherence of sticky material in oral cavity, splitting of	

	shivering, anorexia, indigestion, congestion of eyes, lachrymation, and there is a feeling as if ants are crawling over the body.	eyes, sleeplessness, dislike for everything or restlessness, giddiness, intense desire for cold wind and water.	saliva, vomiting, lassitude, timidity, diminution of digestive fire and strength.	
Clinical presentation	The part where the disease spreads becomes greyish, blackish, reddish. Patient suffer from intense pain which is either pricking, breaking, colic, expansion, contraction, tingling sensation (horripilation) and throbbing sensation. Due to negligence or delay in treatment produces various kinds of afflictions i:e quick bursting, cracking, erupted lesions are small in size, having blackish or reddish discolouration with thin, transparent pink and scanty discharge.	Affected area where the disease located develops coppery, green, yellow, blue, black and red coloured raised skin eruptions having intense burning sensation and breaking pain, suppurating quickly and discharges exudates of similar colour like that of eruptions	The part in which disease spreads becomes oedematous, pale-yellow, slightly reddish and unctuous associated with numbness, stiffness, heaviness and mild pain. Suppuration of the skin eruption is slow, chronic and they appear in large number; the skin over these pustules gets covered with white or pale yellow coloured sticky material. When pustules burst they discharge white, slimy, thready, viscous, knotty and unctuous materials. As the disease advances the area is covered with wounds which are heavy (deep seated), stable, surrounded by capillary network, unctuous and covered with copious smear over skin appear and re-appear. Wounds remain for longer period of time.	
Clinical presentations	Patients suffer from retention of flatus, urine and stool	Eyes, urine and stool becomes deep green and yellowish discolouration and objects appear either green or yellow.	Whitishness of nails, eyes, face, skin, urine, and stool.	
Aggravating and relieving factors	Symptoms aggravate after exposure to etiological factors and alleviates after adoption of etiology opposite to	Symptoms exacerbates after consumption of factors which aggravates *Pitta* and alleviation of sympt-	The etiological factors are non conducive to *Kaphaja Visarpa* and opposite to etiology are conducive to	

	causative ones.	oms takes place after consumption of factors which are opposite to the cause of the disease.	*Kaphaja Visarpa* patient.	
Prognosis *Curable*	*Curable*	*Curable*	*Incurable*	

Differential diagnosis of Āgneya, Granthi and Kardamaka

S.No.	***Āgneya***	***Kardamaka***	***Granthi***
Doṣa	*(Vāta-Pittaja),*	*(Pitta-Kaphaja)*	*(Kapha-Vātaja)*
Clinical presentations	It is characterised by burning sensation all over the body and patient feels as if his body is sprinkled with flaming charcoals associated with vomiting, diarrhoea, fainting, burning sensation, unconsciousness, fever, feeling as if entering into darkness, anorexia, pain in the bones and joints, morbid thirst, indigestion, breaking pain all over the body etc.	It is characterised by cold, fever, heaviness in head, burning sensation, timidity, debility in body parts, sleep, drowsiness, unconsciousness, dislike for food, delirium, diminution of loss of digestive fire, debility, breaking pain in bones, fainting, thirst, adhesion of sticky material inside the *Srotas,* inability to perform normal functions by sensory organs, constant feeling for voiding stool, stretching of extremities, general malaise, restlessness and anxiety. It spreads to *Āmāśaya* in a slow manner and gets localised there.	If person adopts *Rakta* provocative measures along with *Vāta* & *Kapha* aggravating factors leading to vitiation of *Rakta* along with *Vāta* & *Kapha* giving rise to a series of glands in blood vessels, ligaments, muscles and skin. These glands are exceedingly painful, either big or small or elongated or round having reddish discolouration.
Clinical presentations	The part where disease manifested appears like extinguished charcoal or excessive redness and it is surrounded by skin eruptions, which resemble like those caused by burns	The part where disease manifested appears like either red, yellow and pale yellow colour associated with muddy, black, dirty and unctuous. It induces warmthness, heaviness, mild pain which is continuous, swelling and deep seated suppuration.	Patient also suffers from fever, diarrhoea, cough, hiccup, dyspnoea, consumption, unconsciousness, deranged complexion, anorexia, indigestion, salivation, vomiting, fainting, breaking of body parts, sleep, restlessness, malaise etc.
Prognosis	*Agni Visarpa* and *Kardama Visarpa* can be managed by the constant use of general therapeutic measures, provided vital organs are spared and there is no softening of the vessels, ligaments and muscle tissue. Delayed or improper treatment employed to *Agni Visarpa* and *Kardama Visarpa* leads to death of a person like snake poison.		*Granthi Visarpa* without complications can be curable and with complications it is said to be incurable

MCQs

1. *Kunapa Gandhi* is the feature of which *Visarpa*
   a. *Kardama*
   b. *Agni*
   c. *Kshataja*
   d. *Grandhi*
2. Number of types of *Visarpa* as per *Charaka*
   a.*5*
   b. *6*
   c. *8*
   d. *7*
3. Match the predominance of *dosha* in various types of *Visarpa*

Types of *Visarpa*	*Dosha* Predominance
1. Granthi Visarpa	A. Rakta & Pittaja
2. Kardamaka Visarpa	B. Kapha &Vataja
3. Agni Visarpa	C. Kapha &Pittaja
4. Kshataja Visarpa	*D. Vata & Pittaja*

   Choose the correct option
   a. 1 2 3 4 A B C D
   b. 1 2 3 4 B C D A
   c. 1 2 3 4 D C B A
   d. 1 2 3 4 A D B C
4. If abnormal *Pitta* enters *Raktadhatu* manifest which diseases?
   a. *Vishpota*
   b. *Masoorika*
   c. *Visarpa*
   d. *Panduroga*

5.*Kulathasadrisyasphota* is seen in
   a. *Granthi Visarpa*
   b. *Kardamaka Visarpa*
   c. *Agni Visarpa*
   d. *Kshataja Visarpa*
6. *Kurvanti Vistrutamanunnatmashu* is about
   a. *Kushta*
   b. *Prameha*
   c. *Visarpa*
   d. *Vidradi*
7. Which visarpa is known as marmanusari
   a. *Kardama*
   b. *Agni*
   c. *Kshataja*
   d. *Tridoshaja*
8. *Dosha* predominance of *Kshatajavisarpa*
   a. *Rakta & Pittaja*
   b. *Rakta, Vata & Pittaja*
   c. *Rakta&Vata*
   d. *Pittaja*
9. गम्भीरपाकः is the feature of
   a. Kardama
   b. Agni
   c. Kshataja
   d. Grandhi
10. दुष्प्रबोधोऽश्रुतेनिद्रां is the feature
   a. *Kardama*
   b. *Agni*
   c. *Kshataja*
   d. *Tridoshaja*

Answer

1.	a	2.	d	3.	b
4.	c	5.	d	6.	c
7.	b	8.	b	9.	a
10.	b				

CHAPTER
33

Skin Disease

Eczema/ dermatitis

1. The terms eczema and dermatitis are synonymous.
2. Distinctive reaction patterns in the skin cause either acute or chronic eczema.

The acute stage eczema : In the acute stage, oedema of the epidermis (spongiosis) progresses to the formation of intra-epidermal vesicles, which may enlarge and rupture.

The chronic stage eczema : In the chronic stage there is less oedema and vesiculation, but more thickening of the epidermis (acanthosis); this is accompanied by a variable degree of vasodilatation and T-cell lymphocytic infiltration in the upper dermis.

Classification of eczema

- Atopic
- Seborrhoeic
- Discoid
- Irritant
- Allergic
- Asteatotic
- Gravitational
- Lichen simplex
- Pompholyx

Clinical features

1. There are several patterns of eczema; some have identifiable environmental causes, whereas others are more complex. The clinical signs are similar in all types and vary according to the duration of the rash.
2. The features of acute and chronic eczema are as follows:

   The eczema reaction

Acute

- Redness and swelling, usually with ill-defined margins
- Papules, vesicles and, more rarely, large blisters
- Exudation and cracking
- Scaling

Chronic

- May show all of the above, but usually less vesicular and exudative
- Lichenification, a dry leathery thickening with increased skin markings, secondary to rubbing and scratching
- Fissures and scratch marks
- Hypo- or hyperpigmentation

Atopic eczema

1. Atopy is a genetic predisposition to form excessive IgE, which leads to a generalised and prolonged hypersensitivity to common environmental antigens such as pollen and house dust mite.
2. Atopic individuals manifest one or more of a group of diseases that includes asthma, hay fever, food and other allergies, and atopic eczema.

Diagnostic criteria for atopic eczema

Itchy skin and at least three of the following:

- History of itch in skin creases (or cheeks if < 4 yrs)
- History of asthma/hay fever (or in a first-degree relative if < 4 yrs)
- Dry skin (xeroderma)
- Visible flexural eczema (cheeks, forehead, outer limbs if < 4 yrs)
- Onset in first 2 yrs of life

Atopic eczema: distribution and character of rash

Infancy

- Often acute and involves the face and trunk
- Frequent sparing of the napkin area-Childhood
- Backs of the knees, fronts of the elbows, wrists and ankles

Adults

- Face and trunk are involved; lichenification is common

Complications of atopic eczema : Superinfection

Bacterial : (Staphylococcus aureus) most common

Viral

- Herpes simplex virus causes a widespread severe eruption-eczema herpeticum
- Papillomavirus and molluscum contagiosum may be encouraged by use of local corticosteroids

Irritant reactions

- Defective barrier function

Sleep disturbance

- Loss of schooling and behavioural difficulties

Food allergy

- Eggs, cow's milk, protein, fish, wheat and soya may cause an immediate urticarial eruption rather than exacerbation of eczema

Seborrhoeic eczema : This is characterised by a red scaly rash and classically affects the scalp (dandruff), central face, nasolabial folds, eyebrows and central chest. It is due to Pityrosporum ovale infection of the skin. When severe, it may resemble psoriasis. Seborrhoeic eczema is a feature of AIDS, in which case it can be very severe.

Discoid eczema : This common form of eczema is recognised from discrete coin-shaped eczematous lesions, particularly on the limbs of young and elderly men. It can occur in children with atopic eczema and may be difficult to treat.

Irritant eczema

1. Strong irritants elicit an acute reaction at the site of contact, whereas weak irritants most often cause chronic eczema, especially of the hands, after prolonged exposure.
2. Detergents, alkalis, acids, solvents and abrasive dusts are common causes.
3. There is a wide range of susceptibility to weak irritants.
4. Irritant eczema accounts for the majority of occupational cases and work loss.
5. The elderly, those with fair and dry skin, and those with an atopic background are especially vulnerable.

Allergic contact eczema

1. This is due to a delayed hypersensitivity reaction following contact with antigens or haptens
2. Previous exposure to the allergen is required for sensitisation and the reaction is specific to the allergen or closely related chemicals.

Common allergens

Allergen	Present in
Nickel	Jewellery, jean studs, bra clips
Dichromate	Cement, leather, matches
Rubber chemicals	Clothing, shoes, tyres
Colophony	Sticking plaster, collodion
Paraphenylenediamine	Hair dye, clothing
Balsam of Peru	Perfumes, citrus fruits
Neomycin, benzocaine	Topical applications
Parabens	Preservative in cosmetics and creams
Wool	Lanolin, cosmetics, creams
Epoxy resin	Resin adhesives
Allergic contact eczema	

This was caused by the application of an antihistamine cream. The acute eczematous reaction and bilateral periorbital oedema are typical.

Gravitational (stasis) eczema : This occurs on the lower legs and is often associated with signs of venous insufficiency: oedema, red or bluish discoloration, loss of hair, induration, haemosiderin pigmentation and ulceration.

Lichen simplex

- This describes a plaque of lichenified eczema due to repeated rubbing or scratching, as a habit or in response to stress
- Common sites include the nape of the neck, the lower legs and the anogenital area.

Pompholyx

- Recurrent vesicles and bullae occur on the palms, palmar surface of the fingers and soles, and are excruciatingly itchy.

- This form of eczema can occur in atopic eczema and in irritant and contact allergic dermatitis. It can be provoked by heat, stress and nickel ingestion in a nickelsensitive patient but is often idiopathic.

Seborrhoeic eczema : Antipityrosporal agents, such as ketoconazole shampoo and creams, form the basis of treatment, supplemented with weak corticosteroids if needed. Treatments may need to be repeated at intervals.

Irritant eczema : This is treated by the regular use of emollients, protective clothing (e.g. gloves) and avoidance of irritants.

Contact allergic eczema

1. Avoidance of the culprit allergen is the most important intervention; this may entail lifestyle modifications, such as a change in occupation or hobbies.
2. Measures used for irritant eczema are also helpful.

Gravitational eczema

1. Topical corticosteroids should only be applied to eczematous areas and should be avoided on ulcers.
2. Sensitisation to topical antibiotics (neomycin) and preservatives (e.g. chlorocresol) is common. Associated peripheral oedema is treated by elevation of the leg and graded compression bandages.

Investigation of eczema

1. Patch tests are performed in suspected cases of contact allergic dermatitis
2. Prick tests or IgE and specific IgE tests are occasionally performed to support the diagnosis of atopic eczema and to determine specific environmental allergens, e.g. pet dander, horse hair, house dust mite, pollens and foods.
3. Bacterial and viral swabs for microscopy and culture are useful in suspected secondary infection.
4. Bacteria are invariably present, but antibacterial treatment should be reserved for those cases with evidence of clinical infection.
5. Individuals with atopic eczema have an increased susceptibility to herpes simplex virus (HSV), and are at risk of developing a widespread infection, eczema herpeticum. The presence of small punched-out lesions on a background of worsening eczema suggests the possibility of secondary HSV infection.

Urticaria

1. Urticaria refers to an area of focal dermal oedema secondary to a transient increase in capillary permeability.
2. On certain body sites such as the lips or hands, the oedema spreads deeper and is referred to as angioedema.
3. By definition, the swelling lasts less than 24 hours.
4. Acute urticaria may be associated with angioedema of the lips, face, throat and rarely wheezing, abdominal pain, headaches and even anaphylaxis.
5. Whilst severe angioedema can be life threatening due to respiratory obstruction, this is extremely rare in a dermatological context.

Causes of urticaria

Possible allergens, including drugs, should be determined.

Acute and chronic urticaria :

- Autoimmune due to production of antibodies that cross-link the IgE receptor on mast cells
- Allergens (in foods, inhalants and injections)
- Drugs
- Contact, e.g. animal saliva, latex
- Physical, e.g. heat, cold, pressure, sun, water
- Infection, e.g. viral hepatitis, infectious mononucleosis, HIV seroconversion
- Other conditions, e.g. systemic lupus erythematosus (SLE), pregnancy, intestinal parasites
- Idiopathic

Urticarial vasculitis

- Hepatitis B

- SLE
- Idiopathic

Pathogenesis

There is an autoimmune pathogenesis for the most common form of disease

↓

The symptoms and signs of urticaria are due largely to mast cell degranulation with release of histamine and a variety of other vasoactive mediators

↓

More than histamine is clearly involved, as although potent histamine blockers frequently improve the itch of urticaria and the number of weals, they do not abolish them.

↓

Chronic idiopathic urticaria, which is defined by the presence of urticarial episodes for more than 6 weeks

↓

Self-reacting antibodies appear to cause cross-linking of the surface IgE receptor on mast cells with subsequent cellular degranulation.

Clinical history

It is useful to ask to differentiate acute and chronic :

- How long does the individual lesion last? < 24 hours (urticaria)

  > 24 hours (urticarial vasculitis)

- How long has the condition been present? < 6 weeks (acute urticaria)

  > 6 weeks (chronic urticaria)

Distinguishing Urticaria from Urticarial Vasculitits :

1. Knowing the length of time that an individual weal lasts may be of some use in distinguishing urticaria from urticarial vasculitis.
2. Urticarial vasculitis is much less common than urticaria, and many patients are unable to distinguish the development of new weals and disappearance of old ones, from individual weals which persist for more than 1 day.
3. This may be clarified by drawing around a weal with a pen and examining the patient 24 hours later.
4. The physical urticarias can be identified by asking appropriate questions and subsequent challenge testing.

Family history

5. A family history must be sought in cases of angioedema.

Examination

6. Examination may reveal nothing as this is a transient eruption, or may uncover the classical weals which vary from papules to large extensive plaques.

Investigations

- Full *blood count* -including eosinophil count in case of underlying parasites
- *Erythrocyte sedimentation rate* (ESR): may be elevated in cases of vasculitis
- *Urea and Electrolytes, Thyroid and Liver function tests:* may reveal an underlying systemic disorder
- *Total IgE and specific IgE to possible allergens,* e.g shellfish or peanuts
- *Antinuclear factor:* may be positive in chronic urticaria or urticarial vasculitis
- CH50: indicates the level of complement activation; C3 and C4 levels may reveal complement consumption
- *C_1 esterase inhibitor:* may be quantitatively reduced or more rarely functionally deficient, as in hereditary angioedema
- *Skin biopsy:* helpful if urticarial vasculitis is suspected
- *Appropriate challenge test:* to confirm physical urticarias. Frequently no cause can be found for acute episodes, whereas in chronic urticaria an autoimmune pathogenesis will account for the majority of cases.

Psoriasis

1. Psoriasis is a non-infectious, chronic inflammatory disease of the skin, characterised by well-defined erythematous plaques with

silvery scale, with a predilection for the extensor surfaces and scalp, and a chronic fluctuating course.

2. The prevalence is approximately 2% in European populations but may be lower in African and some Asian populations, e.g. Japanese.
3. Psoriasis may start at any age but is unusual before 5 years.
4. There are two epidemiological patterns of psoriasis, sometimes referred to as type 1and type 2.
5. The first has an onset in the teenage and early adult years, often with a family history of psoriasis and an increased prevalence of the HLA group Cw6.
6. The onset of the second is in the fifties or sixties, when a family history is less common and HLA Cw6 is not so prominent.

Aetiology

1. Psoriasis is genetically complex and a large number of genes are thought to be important in its pathogenesis.
2. There is a large familial component.
3. Formal estimates from twin studies suggest a hereditability of around 80%, and in monozygotes perhaps one-third will be concordant for psoriasis.

Factors causing exacerbations of psoriasis

Trauma : Lesions appear in areas of skin damage such as scratches or surgical wounds (Kobner phenomenon) when the condition is erupting

Infection : β-haemolytic streptococcal throat infections often precede guttate psoriasis

Sunlight : UVR rarely

Drugs :

- Antimalarials, (β-adrenoceptor antagonists (β-blockers) and lithium
- 'Rebound' after systemic corticosteroids or potent local corticosteroids are stopped

Emotion : Anxiety

Pathogenesis

There are two key pathophysiological features in psoriatic plaques

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The keratinocytes hyperproliferate with a grossly increased mitotic index and an abnormal pattern of differentiation,

↓

leading to the retention of nuclei in the stratum corneum, not ; normally present as the stratum corneum cells are dead.

↓

There is a large inflammatory cell infiltrate many, it is the first presentation of the disease.

↓

The rash often appears rapidly.

↓

Individual lesions are dropletshaped, seldom over 1 cm in diameter and scaly.

↓

Bouts of guttate psoriasis may clear within a few months but respond well to early treatment with phototherapy

↓

The majority of patients go on to develop plaque psoriasis later in life.

Erythrodermic psoriasis

1. The skin becomes universally red or scaly, or more rarely just red with very little scale.
2. As in other forms of erythroderma, temperature regulation becomes compromised with a danger of either hypothermia or hyperthermia.

Pustular psoriasis

1. There are two varieties of pustular psoriasis.

Generalised form

2. The generalised form is rare but serious.
3. The onset is usually sudden, with large numbers of small sterile pustules erupting on a red base.
4. The patient may rapidly become ill with a pyrexia coinciding with the appearance of new

pustules, and will usually require urgent assessment and hospital admission.

A localised form

5. A localised form, which primarily affects the palms and soles, is more common.
6. This is chronic and comprises small sterile pustules which lie on a red base, and resolve to leave brown macules or scaling.

Clinical features

There are several different forms of psoriasis.

Stable plaque psoriasis :

1. Individual lesions are well demarcated and range from a few millimetres to several centimetres in diameter
2. They are red, with a dry silvery-white scale, which may only be obvious after scraping the surface.
3. The elbows, knees and lower back are commonly involved.
4. Other sites of predilection include:

- Scalp. This is involved in approximately 60% of patients and it is not clear why this is so common.
- Nails. Involvement is common, with thimble pitting', onycholysis (separation of the nail from the nail bed, and subungual hyperkeratosis.
- *Flexures.* Psoriasis involving the natal cleft and submammary and axillary folds is not scaly but red, shiny and symmetrical.
- Palms. Individual plaques may be poorly demarcated and barely erythematous, making this type of psoriasis difficult to differentiate from eczema of the palms.
- Joint pain (psoriatic arthritis) without any visible skin findings
- Conjunctivitis or blepharitis

Auspitz's sign

- It is the appearance of punctate bleeding spots when psoriasis scales are scraped off, named after Heinrich Auspitz.
- It is due to thinning of the epidermal layer overlying the tips of the dermal papillae and blood vessels within the papillae are dilated and tortuous, which bleed readily when the scale is removed.

Guttate psoriasis

This is most commonly seen in children and adolescents, and may follow a streptococcal sore throat.

Lichen Planus

- It is a cell-mediated immune response of unknown origin.
- It may be found with other diseases of altered immunity, such as ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, morphea, lichen sclerosis, and myasthenia gravis.
- A rare type of lichen planus, familial bullous lichen planus, could be gene-related. It may be triggered by diuretics and antimalarials, metal fillings (causing oral lichen planus), stress, and infection.

Clinical presentation

The following may be noted in the patient history:

- Lesions initially developing on flexural surfaces of the limbs, with a generalized eruption developing after a week or more and maximal spreading within 2-16 weeks
- Pruritus of varying severity, depending on the type of lesion and the extent of involvement
- Oral lesions that may be asymptomatic, burning, or even painful
- In cutaneous disease, lesions typically resolve within 6 months (>50%) to 18 months (85%)
- chronic disease is more likely oral lichen planus or with large, annular, hypertrophic lesions and mucous membrane involvement

In addition to the widespread cutaneous eruption, lichen planus can involve the following structures:

- Mucous membranes
- Genitalia
- Nails
- Scalp

The clinical presentation of lichen planus has several variations, as follows:

- Hypertrophic lichen planus
- Atrophic lichen planus
- Erosive/ulcerative lichen planus
- Follicular lichen planus (lichen planopilaris)
- Annular lichen planus
- Linear lichen planus
- Vesicular and bullous lichen planus
- Actinic lichen planus
- Lichen planus pigmentosus
- Lichen planus pemphigoides

Ancillary investigations

- Direct immunofluorescence study reveals globular deposits of immunoglobulin M (IgM) and complement mixed with apoptotic keratinocytes. No imaging studies are necessary.

Distinguishing histopathologic features of lichen planus include the following:

- Hyperkeratotic epidermis with irregular acanthosis and focal thickening in the granular layer
- Degenerative keratinocytes (colloid or Civatte bodies) in the lower epidermis; in addition to apoptotic keratinocytes, colloid bodies are composed of globular deposits of IgM (occasionally immunoglobulin G [IgG] or immunoglobulin A [IgA]) and complement
- Linear or shaggy deposits of fibrin and fibrinogen in the basement membrane zone
- In the upper dermis, a bandlike infiltrate of lymphocytic (primarily helper T) and histiocytic cells with many Langerhans cells

Bullous Pemphigoid

- Bullous pemphigoid is a chronic, inflammatory, subepidermal, blistering disease.
- If untreated, it can persist for months or years, with periods of spontaneous remissions and exacerbations. The disease can be fatal, particularly in patients who are debilitated.

Clinical Presentation- Signs and symptoms

Bullous pemphigoid may present with several distinct clinical presentations, as follows:

- Generalized bullous form: The most common presentation; tense bullae arise on any part of the skin surface, with a predilection for the flexural areas of the skin
- Vesicular form: Less common than the generalized bullous type; manifests as groups of small, tense blisters, often on an urticarial or erythematous base
- Vegetative form: Very uncommon, with vegetating plaques in intertriginous areas of the skin, such as the axillae, neck, groin, and inframammary areas
- Generalized erythroderma form: This rare presentation can resemble psoriasis, generalized atopic dermatitis, or other skin conditions characterized by an exfoliative erythroderma
- Urticarial form: Some patients with bullous pemphigoid initially present with persistent urticarial lesions that subsequently convert to bullous eruptions; in some patients, urticarial lesions are the sole manifestations of the disease
- Nodular form: This rare form, termed pemphigoid nodularis, has clinical features that resemble prurigo nodularis, with blisters arising on normal-appearing or nodular lesional skin
- Acral form: In childhood-onset bullous pemphigoid associated with vaccination, the bullous lesions predominantly affect the palms, soles, and face
- Infant form: In infants affected by bullous pemphigoid, the blisters tend to occur frequently on the palms, soles, and face, affecting the genital areas rarely; 60% of these infant patients have generalized blisters

Ancillary investigations

To establish a diagnosis of bullous pemphigoid, the following tests should be performed:

- Histopathologic analysis: From the edge of a blister; the histopathologic examination demonstrates a subepidermal blister; the inflammatory infiltrate is typically polymorphous, with an eosinophil predominance; mast cells and basophils may be prominent early in the disease course

- Direct immunofluorescence (DIF) studies: Performed on normal-appearing, perilesional skin (see the image below), or nonbullous urticarial skin; significant false-negative rate if skin biopsy is taken from skin of the legs
- Direct immunofluorescence study performed on a perilesional skin biopsy specimen from a patient with bullous pemphigoid detects a linear band of immunoglobulin G deposit along the dermoepidermal junction.
- Indirect immunofluorescence (IDIF) studies: Performed on the patient's serum, if the DIF result is positive
- Indirect immunofluorescence study performed on salt-split normal human skin substrate with the serum from a patient with bullous pemphigoid detects immunoglobulin G class circulating autoantibodies that bind to the epidermal (roof) side of the skin basement membrane.
- DIF tests in patients with bullous pemphigoid usually demonstrate immunoglobulin G (IgG) and complement C3 deposition in a linear band at the dermal-epidermal junction, with IgG in salt-split skin found on the blister roof (epidermal side of split skin).
- IDIF studies document the presence of circulating IgG autoantibodies in the patient's serum that target the skin basement membrane component. Seventy percent of patients with bullous pemphigoid have circulating autoantibodies that bind to split skin.

Pemphigus

1. Pemphigus is an uncommon autoimmune disorder that can present with mucosal symptoms only, usually in the mouth.
2. It is usually due to IgGl and IgG4 auto-antibodies, directed against desmogleins-1 and 3 and causing intra-epidermal blistering, but it can also be drug-induced (e.g. by penicillamine or captopril) and is rarely associated with an underlying malignancy ('para-neoplastic pemphigus').
3. In pemphigus foliaceus, a very superficial form, the autoantibodies are directed solely against desmoglein-1, therefore affecting only the most superficial epidermis..

Clinical features and diagnosis

1. Flaccid blisters occur on the skin, usually on the upper trunk and back.
2. The blisters are superficial; they are fragile and may look like erosions.
3. The Nikolsky sign is positive.
   - It is useful in differentiating between the diagnosis of pemphigus vulgaris or mucous membrane pemphigoid (where the sign is present) and bullous pemphigoid (where it is absent).
   - The Nikolsky sign is dislodgement of intact superficial epidermis by a shearing force, indicating a plane of cleavage in the skin.
4. Mucosal involvement can be severe, increasing the morbidity and risk of mortality.

Investigations

5. Pemphigus can be associated with other autoimmune disorders and full assessment for these is mandatory.
6. In the acute situation, histology from a Tzank smear (taken from the base of the blister with a scalpel) reveals acantholysis, i.e. separation of the epidermal cells.
7. Disease activity can be monitored by measuring the level of circulating autoantibody in serum by indirect immunofluorescence.
8. Investigations should also include full blood count, ESR, urea and electrolytes, liver function tests, chest X-ray and other imaging, particularly if paraneoplastic pemphigus is suspected.

Mycotic skin diseases

- Fungal infections have been known since the 1000 BC., when Ayurveda Samhitas wrote about Kustha.

- Superficial fungal infections have been known since the 5th century BC, when Hippocrates wrote about thrush in children.
- It took hundreds of years before the first scientific proof of infection was made by Agostino Bassi in 1835, who showed that the muscardine disease of the silkworm was caused by a fungus.
- In the following years Audouin from France suggested that some human diseases were caused by the same types of plant parasites (fungi). By the end of the 19th century important microbiological methods, such as obtaining pure cultures of the dermatophytes *Trichophyton* and *Achorion schoenleinii*, were introduced.
- A morphological classification was not established until 1910, when the famous mycologist R. Sabouraud published "*Les teignes*", a monograph based on the standardization of test media and studies on clinical features of skin and hair infections and morphology in cultures. At the beginning of the 20th century different nomenclatural systems were suggested, based on clinical presentation and culture characteristics.

Etiological agents

Anamorph

- *Epidermophyton*, *Microsporum*, and *Trichophyton* are three anamorphic (asexual or imperfect) genera of the anamorphic class Hyphomycetes of the Deuteromycota that comprise the genus Dermatophytes.
- The genera and descriptions of each are listed below-

Trichophyton	:	24 species
Microsporum	:	16 species
Epidermophyton	:	2 species

Trichophyton spp.

Trichophyton tonsurans is the type species. Microconidia are often more numerous than macroconidia and can be pyriform, globose, clavate, stalked, sessile, and borne individually along the edges of the hyphae or in clusters that resemble grapes.

Microsporumspp

Microsporumma fouinii is the type species.

Emmons first classified the macroconidia as spindle- or fusiform-shaped, but the discovery of additional species, such as Microsporum naman, increased the range from obovate (egg-shaped) to cylindrofusi form

Epidermophyton spp.

Epidermophyton floccosum, the sole species of its kind, is anthropophilic, unable to dwell freely in soil, and only sometimes seen to parasitize lesser animals

Teleomorphs

Only a few species of dermatophytes, primarily the geophilic and zoophilic Trichophyton and Microsporum species, are capable of sexual reproduction and the production of ascomata with asci and ascospores.

Ecology

Based on the ecological niches in which they normally reside, dermatophytes have been divided into geophilic, zoophilic, and anthropophilic species. (Spiliopoulou A et al., 2015).

Geophillic

These fungi may infect people and animals and are commonly isolated from the soil where they are linked to keratinous material that is degrading.

Anthropophilic

These dermatophytes are mostly connected to humans and seldom infect other species. Direct contact or indirect transmission by fomites is the two ways these groupings of the organism are spread.

Zoophilic

Zoophilic dermatophytes frequently infect animals and sporadically humans.

This fungus may infect individuals either directly through contact with an infected animal or indirectly through contact with spores.

Ecological classification of dermatophytes .

Geophilic	Anthrophilic	Zoophilic
Trichophyton ajelloi Trichophyton terrestre	*dermophyton floccosum Microsporumaudouinii Microsporumferrugineum Trichophyton concentricum Trichophyton kanei Trichophyton megninii Trichophyton mentagrophytes Trichophyton raubitschekii Trichophyton rubrum Trichophyton schoenleinii Trichophyton soudanense Trichophyton tonsurans*	*Microsporumcanis Microsporumequinum Microsporumpersicolor Microsporumnanum Trichophyton mentagrophyte Trichophyton simii Trichophyton equinum Trichophyton verrucosum*

Epidemiology

WHO estimates that dermatophytes are the major organism responsible for the millions of individuals infected by superficial fungal infections globally, with an estimated lifetime risk of 20–25%. (World Health Organization; 2005).

Although tinea infections are widespread worldwide, they are more frequent in tropical nations like India. Dermatophytosis varies from place to place, person to person, and is caused by a variety of factors, including socioeconomic situations, climate, and population density Dermatophytosis is becoming more common due to a number of causes, including the tropical temperature, urbanisation, overcrowding, shared housing, wearing tightly-fitting clothing, wearing occlusive footwear, participating in sports, and public bathing

While tinea capitis is more frequently found in adolescents and children, tinea cruris and tinea pedis are the most prevalent illnesses in adult males

Clinical Manifestation

Tinea capitis

Tinea capitis, the dermatophyte infection commonly affects scalp and hair, caused by members of the genera Microsporum and Trichophyton, most commonly occurs in children.

It may range from mild, almost subclinical, with slight erythema and a few patchy areas of scaling with dull gray hair stumps to a highly inflammatory reaction with folliculitis, kerion formation, and extensive areas of scarring and alopecia, sometimes accompanied by fever, malaise, and regional lymphadenopathy. Ectothrix, the infection of the hair, appears as a sheath of arthroconidia formed on the outside of the hair shaft.

Pathogens associated with clinical types of Tinea Capitis

Inflammatory	*Microsporumaudouinii, Microsporumcanis, Microsporumgypseum, Microsporumnanum Trichophyton interdigitale, Trichophyton schoenleinii, Trichophyton tonsurans, Trichophyton verrucosum*
Black dot	*T. tonsurans, T. violaceum*
Non-inflammatory	*M. audouinii, M. canis M. ferrugineum, T. tonsurans*
Favus	*T. schoenleinii, T. violaceum, T. mentagrophytes*

Tinea corporis

Tinea corporis (i.e. body ringworm) occurs on the trunk and extremities. The infection may range from mild to severe, commonly appearing as annular erythematous scaly patches with a raised border and central clearing. Varying degrees of itching is present in the lesion.

T. rubrum and E. floccosum are anthropophilic organisms that cause tinea corporis and infect the skin but not the hair, show little inflammation and very little scaling at the edge lead to the chronic lesion. Other dermatophytes also found in tinea capitis, such as M. audouinii, T. schoenleinii, T.

tonsurans and T. violaceum can also cause tinea corporis.

Tinea barbae

Tinea barbae defines the infection of the skin and coarse hairs of the beard and mustache area, which occurs in adult men and hirsute women

Tinea faciei

Tinea faciei involves the infection of the nonbearded areas of the face. Affected patients may have burning and itching, gets aggravated by exposure to sunlight.

Tinea cruris

Tinea cruris, or "jock itch," is a dermatophyte infection of the groin (particularly the proximal medial thighs), perineal and perianal areas, may extend to the buttocks or abdomen, but usually does not involve the scrotum

Tinea pedis

Tinea pedis (Athlete's foot) involves infection of the feet, especially the soles and toe webs. There are three common presentations of tinea pedis. The most common is the interdigital type, which is characterized by itching, maceration, burning sensation, fissuring and peeling in between the toes.

Tinea manuum

Tinea manuum is a dermatophytosis of one or both hands. It is most commonly caused by Trichophyton spp. Palms are typically hyperkeratotic and very dry; fingernails may be involved.

Onychomycosis

Onychomycosis describes fungal infection of the nail caused by a fungus such as dermatophytes, nondermatophyte molds or yeasts. Tinea ungulum refers strictly to dermatophyte infection of the nail, accounts for approximately 90% of cases

1) Distal and Lateral Subungual Onychomycosis (DLSO): it is the most common type (90%), usually caused by T. rubrum. Discoloration, subungual hyperkeratosis and distal onycholysis start at the hyponychium and spreads proximally.
2) Proximal Subungual Onychomycosis (PSO): the dermatophytes invade the nail unit under the proximal nail fold and spread distally. It is usually caused by T rubrum and is usually associated with immunosuppressed conditions, e.g. HIV infection.
3) Superficial White Onychomycosis (SWO): the fungi, mainly 7 mentagrophytes, directly invade the superficial layers of the nail plate but do not penetrate it leading to a white, crumbly nail surface.
4) Total Dystrophic Onychomycosis: It is characterized by thickened, opaque, discolored, broken and dystrophic nails. The nail plate may be separated from the nail bed. Toenails are affected more often than fingernails, and individual nails are sometimes spared.

Tinea imbricata

Tinea imbricata is the chronic infectious manifestation of tinea corporis, characterized by concentric rings of overlapping scales scattered throughout the body.

Tinea incognito

Tinea incognito is mycosis of the skin caused by fungi such as T rubrum, T. mentagrophytes, E. flocosums, M. canis M gypseum and M. jasiel seen in immunocompromised. Symptoms are skin atrophy, telangiectasia and florid growth (combinations of papules, pustules or nodules) on feet, ankle, legs or groin.

Majocchi's Granuloma:

Majocchi's granuloma occurs when dermatophytes invade the dermis and subcutaneous tissues, usually via disrupted hair follicles, as a result of trauma or shaving, largely on the extremities, occasionally on the scalp or face.

Id Reaction:

The id reaction is an acute, local or more generalized, papulovesicular eruption that usually occurs at a site remote from the site of dermatophyte infection. It occurs due to sensitization to circulating antigens and not from direct infection by dermatophytes or other fungal, bacterial, viral or parasitic infections, The id reaction should not be mistaken for a drug reaction.

Investigations

Dermatophytosis is usually diagnosed by a combination of direct microscopic examination, culture and Wood's lamp examination. Biopsy (histopathology) tends to be used mainly when the presentation is unusual.

Wood's Lamp Examination

Wood's lamp is a device that is useful in diagnosis and management of superficial cutaneous fungal infections. Wood's glass consists of barium silicate containing about 9% nickel oxide. It transmits long-wave ultraviolet light with peak of 365 nm that shows characteristic fluorescence produced by some microbial agents. This fluorescence has been used to demonstrate hair infection mainly by microsporum species and one of the trichophyton species. It can also be helpful in detecting some species of dermatophytes, such as M.canis and T. quinckeanum. Not all strains of these organisms exhibit fluorescence,

The chemical substance responsible for positive fluorescence is pteridine. Therefore, other materials containing this substance may also give false-positive fluorescence. The commonest source of error is bluish or purplish fluorescence produced by ointments containing petroleum jelly, scales, serum exudates, lint and dried soap.

Fluorescence produced by different microorganisms under Wood's lamp

Microorganisms	Fluorescence Color
Microsporumaudouinii	Bright-green
Microsporumcanis	Bright-green
Microsporumferrugineum	Blue-green
Microsporumdistortum	Blue-green
Microsporumgypseum	Dull-yellow
Trichophyton schoenleinii	Dull-green

Laboratory Diagnosis

The diagnosis of dermatophytosis is based on combination of clinical observation and laboratory investigations depend on demonstration of causative pathogen in skin-scraping by microscopy, culture, serological and molecular test

Collection and Transport of Specimens

Dermatophytes shows radial growth and the centers of infected skin patches consist of the older and the poorly viable material. The clinical specimen like skin scales near the advancing edges of the rings, nail clipping or curettings from dystrophic subungual debris and hair stubs are usually examined. Firstly, the lesion is lightly disinfected with alcohol in gauze and then it is scraped from center to edge, crossing the lesion margin by using a sterile scalpel blade or equivalent.

If lesions consist of vesicles or bullae, then top of the vesicle or bulla should be collected as a sample. In tinea capitis and finea barbae, basal root portion of the hair is best for direct microscopy and culture. From nails, close clippings of the whole nail end are taken from an appropriate site depending on the type of nail infection.

Direct Examination

Despite false negatives in 5 to 15% of cases in ordinary practice, the microscopic examination of KOH wet mounts of keratinous material is very simple and reliable screening technique. Scrapings may be mounted for direct examination in 10% KOH for skin scrapings and 20-40% KOH for hair and nail samples.

In the KOH wet mount, fungus is seen as branching hyaline mycelia, which frequently show arthrospores production particularly in the infected skin appendages like hair. Ectothrix and endothrix types of infections are distinguished in wet mount and arrangement of arthrospores can be made out. The demonstration of fungus in nails may be difficult and may be possible only after keeping clippings in KOH for overnight.

Fungal Culture

The clinical specimens should be inoculated on fungal culture media irrespective of the findings of direct examination. Culture is essential and valuable adjunct to direct microscopy.

Dermatophytes can also grow easily on Sabouraud's Dextrose Agar with antibiotics or cycloheximide and the media should be incubated at three temperatures i.e. 25ãC, 30ãC and 37ãC. The growth is relatively slow and usually takes about 10 days to three weeks time

The typical isolates of common dermatophytes that can be identified directly from primary isolation media like Sabouraud's Dextrose Agar and Potato Glucose or Potato Flake Agar on the basis of identification characteristics like colony pigmentation, texture, and growth rate and distinctive morphological structures, such as microconidia, macroconidia, spirals, pectinate branches, pedicels, and nodular organs.

Culture is a precious accessory of microscopy which is crucial in all infections for treatment with systemic prescription.

BCP-milk solids-glucose agar is used to differentiate dermatophytes such as T. rubrum, T. mentagrophytes, T. soudanense, T. megninti, M. persicolor and M. equinum, on the basis of catabolite domination by glucose and divergence of releasing ammonium ions from casein.

Littman ox gall agar as restrictive media is preferred to diagnose a non dermatophyte infection.

Casamino acids-erythritol-albumin medium was firstly used by Fischer and Kane. This medium contains balanced egg albumin, which reduces yeasts such as C. albicans and is an extremely usable medium for isolating dermatophytes from heavily contaminated samples.

Bromocresol purple (BCP) casein-yeast extract agar is designed for the rapid recognition of microcolonies of T. verrucosum though all dermatophytes are able to grow on this medium.

Biochemical and Physiological Tests-

Urease Activity

Some dermatophytes are able to hydrolyse high concentrations of urea. This test is useful to differentiate T. rubrum (urease negative) from T.mentagrophytes var, interdigitale (urease positive).

In vitro Hair Perforation

Some dermatophytes are able to penetrate and invade hair shaft by producing specialized perforating organs in vitro, while other species attack the hair by simple peripheral erosions

Mating Test

Dermatophytes are heterothallic species. In order to obtain a sexual reproduction, it is necessary to confront strains from the same species with complementary (+ and -) isolates. To observe the teleomorph (Arthroderma genus) state of Microsporum or Trichophyton species, reference strains with complementary polarity are required. Takashio's or De Vroey's (Guiauteabyssinica seeds) media, that are poor culture media stimulating the sexual conidiation of fungi, can be used.

Immunodiagnosis

The skin test with dermatophytic antigen, trichophytin and serological tests are important for diagnosis of dermatophytosis. The trichophytin is crude extract from dermatophytes, producing positive delayed-type hypersensitivity, tuberculin-like response in most of the adults.

Molecular diagnosis of dermatophytosis

Nucleic acid-based molecular methods are increasingly being employed for rapid and specific identification of the fungi as well as for the detection of etiological agent directly from the clinical specimen in the clinical microbiological laboratory

Leprosy

1. Leprosy (Hansen's disease) is a chronic granulomatous disease affecting skin and nerves, caused by *Mycobacterium leprae,* a slow-growing mycobacterium which cannot be cultured in vitro.
2. The clinical manifestations are determined by the degree of the patient's cellmediated immunity (CMI) towards M. *leprae*.
3. High levels of CMI with elimination of leprosy bacilli produce tuberculoid leprosy, whereas absent CMI results in lepromatous leprosy.
4. The complications of leprosy are due to nerve damage, immunological reactions and bacillary infiltration.

5. Leprosy patients are frequently stigmatised and using the word 'leper' is inappropriate.

Epidemiology and transmission

1. Some 4 million people have leprosy and around 750 000 new cases are detected annually.
2. 70% of the world's leprosy patients live in India, with the disease endemic in Brazil, Indonesia, Mozambique, Madagascar, Tanzania and Nepal.

Spread of infection

3. Untreated lepromatous patients discharge bacilli from the nose.
4. Infection occurs through the nose, followed by haematogenous spread to skin and nerve.
5. The incubation period is 2-5 years for tuberculoid cases and 8-12 years for lepromatous cases.
6. Leprosy incidence peaks at 10-14 years, and is more common in males and in those with close household exposure to leprosy cases.

Pathogenesis

M. leprae has a predilection for infecting Schwann cells and skin macrophages.

↓

In tuberculoid leprosy, effective CMI controls bacillary multiplication ('paucibacillary') but organised epithelioid granulomas are formed.

↓

In lepromatous leprosy, there is abundant bacillary multiplication ('multibacillary'), e.g. in Schwann cells and perineurium.

↓

Between these two extremes is a continuum, varying from patients with moderate CMI (borderline tuberculoid) to patients with little cellular response (borderline lepromatous).

↓

Associated with immunological reactions to the infection occur as the immune response develops and the antigenic stimulus from the bacilli varies

↓

particularly in borderline patients.

↓

Delayed hypersensitivity reactions produce type 1 (reversal) reactions, while immune complexes contribute to type 2 (erythema nodosum leprosum) reactions.

↓

HIV/leprosy co-infected patients have typical lepromatous and tuberculoid leprosy skin lesions and typical leprosy histology and granuloma formation.

↓

Surprisingly, even with low circulating CD4 counts, tuberculoid leprosy may be observed and there is not an obvious shift to lepromatous leprosy.

Clinical features

Cardianal features of leprosy

- Skin lesions, typically anaesthetic at tuberculoid end of spectrum
- Thickened peripheral nerves
- Acid-fast bacilli on skin smears or biopsy

Clinical characteristics of the polar forms of leprosy

Clinical and tissue specific features	Lepromatous	Tuberculoid
Skin and nerves		
Number and distribution	Widely disseminated	One or a few sites asymmetrical
Skin lesions		
Definition		
Clarity of margin	Poor	Good
Elevation of margin	Never	Common
Colour		
Dark skin	Slight	Marked
hypopigmentation	hypopigmentation	
Light skin	Slight erythema	Coppery or red

Surface	Smooth, shiny	Dry, scaly
Central healing	None	Common
Sweat and hair growth	Impaired late	Impaired early
Loss of sensation	Late	Early and marked
Nerve enlargement and damage	Late	Early and marked
Bacilli (bacterial index)	Many (5 or 6+)	Absent (D)
Natural history	Progressive	Self-healing
Other tissues	Upper respiratory mucosa, eye, testes, bones, muscle	None
Reactions	Immune complexes (type 2)	Cell-mediated (type 1)

Leprosy reactions

- *Type 1 (reversal) reactions* : These occur in 30% of borderline patients (BT, BB or BL) and are delayed hypersensitivity reactions. Skin lesions become erythematous. Peripheral nerves become tender and painful, with sudden loss of nerve function. These reactions may occur spontaneously, after starting treatment and also after completion of multidrug therapy.
- *Type 2 (erythema nodosum leprosum-ENL) reactions* : These are partly due to immune complex deposition and occur in BL and LL patients who produce antibodies and have a high antigen load. They manifest with malaise, fever and crops of small pink nodules on the face and limbs. Iritis and episcleritis are common. Other signs are acute neuritis, lymphadenitis, orchitis, bone pain, dactylitis, arthritis and proteinuria. ENL may continue intermittently for several years.

Reactions in leprosy

	Lepra reaction type 1 (reversal)	Lepra reaction type 2 (erythema nodosum leprosum, ENL)
Mechanism	Cell-mediated hypersensitivity	Immune complexes
Clinical features	Painful tender nerves, loss of function Swollen skin lesions New skin lesions lymphadenitis Fever, oedema	Tender papules and nodules; may ulcerate Painful tender nerves, loss of function Iritis, orchitis, myositis,
Management	Prednisolone 40 mg, reducing over 3-6 months'	Moderate: prednisofone 40 mg daily Severe: thalidomidez or prednisolone 40-80 mg daily, reducing over 1-6 months; local if eye involved

Borderline cases

1. In borderline tuberculoid (BT) cases, skin lesions are more numerous than in tuberculoid (TT) cases, and there is more severe nerve damage and a risk of type 1 reactions.
2. In borderline leprosy (BB) cases, skin lesions are numerous and vary in size, shape and distribution; annular lesions are characteristic and nerve damage is variable.
3. In borderline lepromatous (BL) cases, there are widespread small macules in the skin and widespread nerve involvement; both type 1 and type 2 reactions occur.
4. Pure neural leprosy (i.e. without skin lesions) occurs principally in India and accounts for 10% of patients.
5. There is asymmetrical involvement of peripheral nerve trunks and no visible skin lesions.
6. On nerve biopsy all types of leprosy have been found.

Investigations

1. The diagnosis is clinical, made by finding a cardinal sign of leprosy and supported by finding acid-fast bacilli in slit-skin smears or typical histology in a skin biopsy.
2. Slit-skin smears are obtained by scraping dermal material on to a glass slide. The smears are then stained for acid-fast bacilli, the number

counted per high-power field and a score derived on a logarithmic scale (0-6): the bacterial index (BI).

3. Smears are useful for confirming the diagnosis and monitoring response to treatment.
4. Neither serology nor PCR testing for M. *leprae* DNA is sensitive or specific enough for diagnosis.

Vitiligo

- Vitiligo is an acquired pigmentary disorder of the skin that is characterized by circumscribed, depigmented macules and patches.
- The condition is frequently associated with disorders of autoimmune origin, with thyroid abnormalities being the most common.

Clinical presentation -Signs and symptoms

Vitiligo lesions are characterized as follows:

- White or depigmented macules and patches
- Usually well demarcated
- Round, oval, or linear in shape
- Borders may be convex
- Range from millimeters to centimeters in size
- Enlarge centrifugally over time at an unpredictable rate
- Initial lesions occur most frequently on the hands, forearms, feet, and face, favoring a perioral and periocular distribution.

Clinical variants

- Trichrome vitiligo
- Marginal inflammatory vitiligo
- Quadrichrome vitiligo
- Koebner phenomenon: Development of vitiligo in sites of specific trauma, such as a cut, burn, or abrasion.

Ancillary investigation

- Although the diagnosis of vitiligo generally is made on the basis of clinical findings, biopsy is occasionally needed for differentiating vitiligo from other hypopigmenting or depigmenting disorders.
- Microscopic examination of involved skin shows a complete absence of melanocytes in association with a total loss of epidermal pigmentation.
- Superficial perivascular and perifollicular lymphocytic infiltrates may be observed at the margin of vitiliginous lesions, consistent with a cell-mediated process destroying melanocytes.

Other documented histologic findings include the following:

- Degenerative changes in keratinocytes and melanocytes in the border lesions and adjacent skin
- Increased numbers of Langerhans cells
- Epidermal vacuolization
- Thickening of the basement membrane
- Loss of pigment and melanocytes in the epidermis is highlighted by Fontana-Masson staining and immunohistochemistry testing.

Cellulitis

- The term cellulitis commonly is used to indicate a nonnecrotizing inflammation of the skin and subcutaneous tissues, usually from acute infection.
- Cellulitis usually follows a breach in the skin, although a portal of entry may not be obvious; the breach may involve microscopic skin changes or invasive qualities of certain bacteria.

Etilogy-Clinical presentation -Signs and symptoms

Nonpurulent cellulitis is associated with the four cardinal signs of infection, as follows :

- Erythema
- Pain
- Swelling
- Warmth

Physical examination findings that suggest the most likely pathogen include the following:

- Skin infection without underlying drainage, penetrating trauma, eschar, or abscess is most likely caused by streptococci
- Staphylococcus aureus, often community-

acquired MRSA, is the most likely pathogen when these factors are present.

- Violaceous color and bullae suggest more serious or systemic infection with organisms such as Vibrio vulnificus or Streptococcus pneumoniae

The following findings suggest severe infection:

- Malaise, chills, fever, and toxicity
- Lymphangitic spread (red lines streaking away from the area of infection)
- Circumferential cellulitis
- Pain disproportionate to examination findings

Indications for emergent surgical evaluation are as follows :

- Violaceous bullae
- Cutaneous hemorrhage
- Skin sloughing
- Skin anesthesia
- Rapid progression
- Gas in the tissue
- Hypotension

Ancillary investigations

Generally, no workup is required in uncomplicated cases of cellulitis that meet the following criteria:

- Limited area of involvement
- Minimal pain
- No systemic signs of illness (eg, fever, altered mental status, tachypnea, tachycardia, hypotension)
- No risk factors for serious illness (eg, extremes of age, general debility, immunocompromise)

The Infectious Disease Society of America (IDSA) recommends the following blood tests for patients with skin or soft tissue infection (SSTI) who have signs and symptoms of systemic toxicity:

- Blood cultures
- CBC with differential
- Levels of creatinine, bicarbonate, creatin phosphokinase, and C-reactive protein (CRP)

Blood cultures should also be done in the following circumstances :

- Moderate to severe disease (eg, cellulitis complicating lymphedema
- Cellulitis of specific anatomic sites (eg, facial and especially ocular areas)
- Patients with a history of contact with potentially contaminated water
- Patients with malignancy who are receiving chemotherapy
- Neutropenia or severe cell-mediated immunodeficiency
- Animal bites

Other tests to consider are as follows:

- Mycologic investigations are advisable if recurrent episodes of cellulitis are suspected to be secondary to tinea pedis or onychomycosis
- Creatinine levels help assess baseline renal function and guide antimicrobial dosing

Imaging studies

- Ultrasonography may play a role in the detection of occult abscess and direction of care
- Ultrasonographic-guided aspiration of pus can shorten hospital stay and fever duration in children with cellulitis
- If necrotizing fasciitis is a concern, CT imaging typically is used in stable patients; MRI can be performed, but MRI typically takes much longer than CT scanning
- Strong clinical suspicion of necrotizing fasciitis should prompt surgical consultation without delay for imaging

Aspiration, Dissection, and Biopsy

- Needle aspiration should be performed only in selected patients or in unusual cases, such as in cases of cellulitis with bullae or in patients who have diabetes, are immunocompromised, are neutropenic, are not responding to empiric therapy, or have a history of animal bites or immersion injury .
- Aspiration or punch biopsy of the inflamed area may have a culture yield of 2-40% and is of limited clinical value in most cases

- Gram stain of aspiration or biopsy specimens has a low yield and is unnecessary in most cases, unless purulent material is draining or bullae or abscess is present; however, Gram stain and culture after incision and drainage of an abscess yields positive results in more than 90% of cases
- Dissection of the underlying fascia to assess for necrotizing fasciitis may be determined by surgical consultation or indicated following initial evaluation and imaging studies
- Skin biopsy is not routine but may be performed in an attempt to rule out a noninfectious entity

Hospital admission

The IDSA recommends considering inpatient admission in patients with hypotension and/or the following laboratory findings:

- Elevated creatinine level
- Elevated creatine phosphokinase level (2-3 times the upper limit of normal)
- CRP level >13 mg/L (123.8 mmol/L)
- Low serum bicarbonate level
- Marked left shift on the CBC with differential

MCQs

1. Leonine facies seen in

a. Tuberculosis b. Leprosy
c. Amyloid d. AIDS

2. In which skin disorder do T-cells induce a sudden increase in cytokines leading to hyperplasia of skin cells resulting in raised skin plaques?

a. dermatitis b. eczema
c. psoriasis d. impetigo

3. Which among the following is not correct regarding severe cellulitis

a. Elevated creatinine level
b. Elevated creatine phosphokinase level (2-3 timesthe upper limit of normal)
c. CRP level &gt;13 mg/L (123.8 mmol/L)
d. High serum bicarbonate level

4. Which of the following is the most common site of cellulitis?

a. Face b. Hand
c. Leg d. Arm

5. Which of the following pathogens is most likely responsible for a skin infection with underlying drainage, penetrating trauma, eschar, or abscess?

a. Staphylococcus aureus
b. Vibrio vulnificus
c. Streptococcus pneumoniae
d. Pseudomonas aeruginosa

Answer

1.	b	2.	c	3.	d
4.	c	5.	a		

CHAPTER 34

Galaganda

स्वरूप (Nature of Diseases)

निबद्धः श्वयथुर्यस्य मुष्कवल्लम्बते गले।
महान् वा यदि वा ह्रस्वो गलगण्डं तमादिशेत्॥
S.S.Ni. 11/31

The disease *Galagaṇḍa* is characterised by swelling which hangs over the region of *gala* like *muṣka*, which may be either big or small in size.

गलस्यपार्श्वेगलगण्डएकः॥ *C.S.Ci. 12/79*

Single swelling over the side of *gala* is the clinical feature of *Galagaṇḍa*.

पवनश्लेष्ममेदोभिर्गलगण्डो भवेद्बहिः।
वर्धमानः स कालेन मुष्कवल्लम्बते निरुक्॥
A.H.Utt. 21/53

Impaired *vāta, kapha* and *medas* causes swelling in the outer aspect of *gala* and gradually increases its size associated with severe pain and hangs like mushka.

निदान (Nidāna)

मात्स्यमाहिषवाराहपिशितामकमूलकम्।
माषसूपदधिक्षीरसुक्तेक्षुरसफाणितम्॥
अवाक्शय्यां च भजतो द्विषतो दन्तधावनम्।
धूमच्छर्दनगण्डूषानुचितं च सिराव्यधम्॥
क्रुद्धाः श्लेष्मोल्बणा दोषाः कुर्वन्त्यन्तर्मुखं गदान्।
A.H.Utt. 21/1-2

Excessive intake of meat of *matsya* (fish), *māhiṣa* (buffalow) and *vārāha* (pig), unripened *mūlaka* (Radish), *māṣasūpa* (black gram soup), buttermilk, milk, *ikṣurasa* (sugar cane juice), and *phāṇita* (inspissated juice of the sugarcane), improper sleeping position, avoidance of toothbrush, improper *dhūmapāna* (smoke), *vamana* (emesis), *gaṇḍūṣa* (gargle) and *sirā vyadhanam* (vein puncture) therapeutics leading to exacerbation and vitiation of *kaphādi doṣa* and manifest diseases of *mukha*.

संप्राप्ति (Samprāptī)

यस्य श्लेष्मा प्रकुपितो गलबाह्येऽवतिष्ठते।
शनैः संजनयेच्छोफं गलगण्डोऽस्य जायते॥
यस्य श्लेष्मा प्रकुपितस्तिष्ठत्यन्तर्गले स्थिरः।
आशु संजनयेच्छोफं जायतेऽस्य गलग्रहः॥
C.S.Su. 18/21-22

If the aggravated *Kapha* is located *Galabāhya* (outside the throat), it causes gradual swelling called as *Galagaṇḍa*, it is chronic in nature.

वातः कफश्चैव गले प्रवृद्धौ मन्ये तु संसृत्य तथैव मेदः।
कुर्वन्ति गण्डं क्रमशः स्वलिङ्गैः समन्वितं तं गलगण्डमाहुः॥
S.S.Ni. 11/23

The aggravated *Vāta* and *kapha* amalgamate with the *medas* (fat) and produce swelling in the neck and its sides. The manifestation of symptoms of the *doṣas* develop gradually. This disease is known and *Galagaṇḍa*.

Intake of etiological factors
↓
Vitiation and aggravation of *Vāta, Kapha* and *Meda*
↓
Reaches *Gala* (Neck)
↓
Amalgamation of *doṣās* and *dūṣyās* in the *Gala* (neck region)
↓
Produces swelling in Duter Ospect of *Gala*
↓
Gradually increases its size associated with sever pain & hangs like *Muṣka*
↓
Galagaṇḍa

भेद (Bhedā)

वातज कफज मेदोज....

Galagaṇḍa is classified into three varieties namely *Vātaja, Kaphaja* and *Medoja. Pittaja* type of *Galagaṇḍa* is not mentioned due to specific *guṇās* of *Pitta*.

वातज गलगण्ड (Vātaja Galagaṇḍa)

कृष्णोऽरुणो वा तोदाढ्यः सा वातात्कृष्णराजिमान् ।
वृद्धस्तालुगले शोषं कुर्याच्च विरसास्यताम् ॥
A.H.Utt. 21/54

Vātaja Galagaṇḍa is characterised by the black or reddish colour swelling associated with pricking type of pain, appearance of black colour veins in the neck and aggravated *vāta* dries up the *tālu* (palate) and *gala* (neck-throat) associated with bitterness of mouth.

कफज गलगण्ड (Kaphaja Galagaṇḍa)

स्थिरः सवर्णः कण्डूमाञ् शीतस्पर्शो गुरुः कफात् ।
वृद्धस्तालुगले लेपं कुर्याच्च मधुरास्यताम् ॥
A.H.Utt. 21/55

Kaphaja Galagaṇḍa develops due to exacerbation of *kapha* and it is characterised by swelling over the gala which is immovable and resembles the colour of the body associated with itching, cold to touch and feeling of heaviness. It also causes coating like feeling in the *tālu* and *gala* along with sweetishness in the mouth.

मेदज गलगण्ड (Medaja Galagaṇḍa)

मेदसः श्लेष्मवृद्धातिवृद्ध्योः सोऽनुविधीयते ।
देहं वृद्धश्च कुरुते गले शब्दं स्वरेऽल्पताम् ॥
A.H.Utt. 21/56

Clinical features of *Medaja Galagaṇḍa* are similar to the features of *Kaphaja Galagaṇḍa*. It increases and decreases its size in proportion with the body size and due to its excessiveness it causes *alpasvara*.

साध्यासाध्यता (Sādhyāsādhyatā)

साध्याः स्मृताः पीनसपार्श्वशूलकासज्वरच्छर्दियुतास्त्वसाध्यः ॥
C.S..Ci. 12/79

कृच्छ्राच्छ्वसन्तं मृदुसर्वगात्रं संवत्सरातीतमरोचकार्तम् ।
क्षीणं च वैद्यो गलगण्डिनं तं भिन्नस्वरं चैव विवर्जयेत्तु ॥
S.S.Ni. 11/30

Galagaṇḍa is considered as curable if there is no association of *Pīnasa, Pārsvasūla, Kāsa, Jvara* and *Chardi*. If it is associated with *Śvāsa, Mṛdutā* in *gātra*, persists for more than 1 year, *Arocaka, Daurbalya* and *Bhinnasvaram* such cases of *galagaṇḍa* should not be treated.

गलगण्डः स्वरभ्रंशी कृच्छ्रोच्छ्वासोऽतिवत्सरः ।
A.Utt. 21/69

Galagaṇḍa which is associated with *Svarabhraṁsa, Kṛchrasvāsa* and lasts for more than a year are considered to be as *asādhya*.

Thyroid enlargement

1. Palpable thyroid enlargement is common, affecting about 5% of the population.
2. Multinodular goitres and solitary nodules sometimes present with acute painful enlargement due to haemorrhage into a nodule.

Causes of thyroid enlargement

Diffuse goitre

- Simple goitre
- Transient thyroiditis
- Hashimoto's thyroidis
- Dyshormonogenesis'
- Graves' disease
- Drugs
- Infiltrative
- Iodine, amiodarone, lithium
- Amyloidosis, sarcoidosis etc
- Iodine deficiency (endemic
- Riedel's thyroiditis
- Suppurative thyroiditis
- Multinodular goitre
- Solitary nodule
- Simple cyst
- Medullary cell
- Colloid nodule carcinoma
- Follicular adenoma
- Anaplastic carcinoma
- Papillary carcinoma
- Lymphoma
- Follicular carcinoma
- Metastasis

Diffuse goitre

1. In the absence of thyrotoxicosis or hypothyroidism a diffuse goitre rarely needs further investigation or treatment unless it is very large and causing cosmetic symptoms or compression of other local structures (resulting in stridor or dysphagia).
2. The presence of autoantibodies may supports the diagnosis of Graves' disease or Hashimoto's thyroiditis, while their absence in a younger patient suggests a simple goitre.
3. Thyroxine therapy is sometimes justified in an attempt to shrink the goitre.

Multinodular goitre

1. It is usually necessary to confirm the clinical diagnosis using 99m Tc scintigraphy or ultrasonography.
2. Sometimes one of the nodules is much larger than any other (a 'dominant' nodule).
3. If such a nodule is 'cold' on isotope scanning, it may be investigated in the same way as a truly solitary nodule since the risk of malignancy is not negligible in such lesions.

Solitary thyroid nodule

1. It is important to determine whether the nodule is benign or malignant.
2. It is rarely possible to make this distinction on clinical grounds alone, although the presence of cervical lymphadenopathy increases the likelihood of malignancy.
3. A solitary nodule presenting in childhood or adolescence, particularly if there is a past history of head and neck irradiation, or one presenting in the elderly should heighten suspicion of a primary thyroid.

MCQs

1. आहारपरिणामान्तेभूयश्चलभतेबलम is clinical feature of
   a. व्यपेताहिक्का (Vyapeta Hikk
1. Which among the following is not a type of *Galaganda*
   a. *Vataja* b. *Paittika*
   c. *Kaphaja* d. *Medoja*
2. *Mushkavatlambhate* is the feature of
   a. *Galaganda* b. *Apachi*
   c. *Grandhi* d. *Vriddhi*
3. *Dehanurupakshayavridhiyukta* is
   a. *Kshayajakasa*
   b. *Medojagalaganda*
   c. *Rajayakshma*
   d. *Apachi*

Answer

1.	c	2.	a	3.	b

CHAPTER

35

Thyroid Disorders

Hypothyroidism

The vast majority of cases hypothyroidism results from an intrinsic disorder of the thyroid gland (primary hypothyroidism).

Causes of hypothyroidism

1. Causes Autoimmune
   Hashimoto's thyroiditis
   Spontaneous atrophic hypothyroidism
   Graves' disease with TSH receptorblocking antibodies
2. Iatrogenic
   Radioactive iodine ablation
   Thyroidectomy
3. Drugs
   Carbimazole, methimazole, propylthiouracil
   Amiodarone
   Lithium
4. Transient thyroiditis
   Subacute (de Quervain's) thyroiditis
   Post-partum thyroiditis
5. Iodine deficiency
   e.g. in mountainous regions
6. Congenital
   Dyshormonogenesis
   Thyroid aplasia
7. Infiltrative
   Amyloidosis, Riedel's thyroiditis, sarcoidosis etc.
8. Secondary hypothyroidism TSH deficiency

Incidence

↓

Women are affected approximately six times more frequently than men

Clinical features of Hypothyroidism

Symptoms	Signs
Common	
Weight gain	Weight gain
Cold intolerance	
Fatigue, somnolence	
Dry skin	
Dry hair	
Menorrhagia	
Constipation	Hoarse voice
Hoarseness	Facial features:
Carpal tunnel syndrome	Purplish lips
Alopecia	Malar flush
Aches and pains	Periorbital oedema/ myxoedema
Muscle stiffness	Loss of lateral eyebrows
Deafness	Anaemia
Depression	Carotenaemia
Infertility	Erythema ab igne (Granny's tartan)
	Bradycardia
	Hypertension
	Delayed relaxation of tendon reflexes
	Dermal myxoedema
Rare	
Psychosis (myxo-edema madness)	Ileus Ascites
Galactorrhoea	Pericardial and pleural effusions
Impotence	Cerebellar ataxia
Gynaecomastia	Myotonia

Investigations

1. Serum T4 is low and TSH is elevated, usually in excess of 20 mU/ L.
2. Measurements of serum T_3 are unhelpful since they do not discriminate reliably between euthyroidism and hypothyroidism.
3. The rare condition of secondary hypothyroidism is caused by failure of TSH

secretion in a patient with hypothalamic or anterior pituitary disease.This is characterised by a low Serum T4 but TSH may be low; normal or even slightly elevated.

4. Serum enzymes Raised creatine kinase, aspartate aminotransferase, lactate dehydrogenase (LDH)
5. Hypercholesterolaemia
6. Anaemia
   Normochromic normocytic or macrocytic
7. Hyponatraemia
8. The electrocardiogram (ECG) classically demonstrates sinus bradycardia with low-voltage complexes and ST segment and T wave abnormalities.
9. Measurement of thyroid peroxidase antibodies is helpful

Hyperthyroidism/Thyrotoxicosis

Definition

Thyrotoxicosis describes a constellation of clinical features arising from elevated circulating levels of thyroid hormone.

Aetiology

The most common causes

1. Graves' disease
2. Multinodular goitre
3. Autonomously functioning thyroid nodules (toxic adenoma)
4. Thyroiditis is more common in parts of the world where relevant viral infections occur, such as North Amcrica.

Less common

- Drugs (e.g. amiodarone)
- Radiographic contrast media
- Iodine prophylaxis programme
- TSH-secreting pituitary adenoma
- Follicular carcinoma - metastases

Clinical features of Thyrotoxicosis

Symptoms	Signs
Common	
Weight loss despite normal or increased appetite	Weight loss
Heat intolerance	Palmar erythema
Palpitations	Sinus tachycardia
Dyspnoea	Lid retraction, lid lag
Irritability, emotional lability	
Fatigue	
Sweating	
Tremor	
Less common	
Osteoporosis (fracture, loss of height)	Goitre with bruit
Diarrhoea, steatorrhoea	Atrial fibrillation
Angina	Systolic hypertension
Ankle swelling	Increased pulse pressure
Anxiety, psychosis	Cardiac failure
Muscle weakness	Hyper-reflexia
Periodic paralysis (predominantly in Chinese)	Ill-sustained clonus
Pruritus	Proximal myopathy
Alopecia	Bulbar myopathy
Amenorrhoea/oligomenorrhoea	
Infertility, spontaneous abortion	
Loss of libido, impotence	
Excessive lacrimation	
Rare	
Vomiting	Lymphadenopathy
Apathy	Spider naevi
Anorexia	Onycholysis
Exacerbation of asthma	Pigmentation/ Gynaecomastia

Investigations

1. The first-line investigations are serum T3, T4 and TSH.
2. In most patients serum T3 and T4 are both elevated but T_4 is in the upper part of the normal range and T3 raised (T3 toxicosis) in about 5%.
3. Serum TSH is undetectable in primary thyrotoxicosis but values can be raised in the very rare syndrome of secondary thyrotoxicosis caused by a TSH-producing pituitary adenoma.
4. measurement of receptor antibodies (TRAb), elevated in Graves' disease
5. Isotope scanning
6. An ECG may demonstrate sinus tachycardia or atrial fibrillation.
7. Radio-iodine uptake tests measure the proportion of isotope which is trapped in the whole gland, but have been largely superseded by 99m technetium scintigraphy scans which also indicate trapping, are quicker to perform with a lower dose of radioactivity, and provide a higher-resolution image.
8. Serum enzymes : Raised alanine amino-transferase, y-glutamyl transferase (GGT), and alkaline phosphatase from liver and bone
9. Raised bilirubin
10. Mild hypocalcaemia
11. Glycosuria

    Associated diabetes mellitus

    'Lag storage' glycosuria

MCQs

1. Which of the following cellular processes increase in response to thyroid-stimulating hormones?

a. No Synthesis of thyroglobulin proteins & Incorporation of iodide ions into tyrosine molecules of thyroglobulin

b. No Incorporation of iodide ions into tyrosine molecules of thyroglobulin

c. No Uptake of iodide into the follicular cells

d. Uptake of iodide into the follicular cells, Synthesis of thyroglobulin proteins & Incorporation of iodide ions into tyrosine molecules of thyroglobulin

2. Which of the following is not a characteristic feature of Grave disease?

a) Increased total and free T4

b) Suppressed plasma TSH

c) Exophthalmos

d) Decreased thyroid radioactive iodine uptake

3. Which of the following form is the active form of thyroid hormone?

a. T3

b. T4

c. rT3

d. TSH

4. The subclinical hyperthyroidism is characterized by________________

a. Increased TSH and normal free T3 & free T4 levels in the blood

b. Increased TSH and decreased free T3 & free T4 levels in the blood

c. Decreased TSH and normal free T3 & free T4 levels in the blood

d. Decreased TSH and increased free T3 and free T4 levels in the blood

5. A patient presents with tachycardia and heat intolerance and is suspected of Graves' disease. Which of the following is not a characteristic feature of Grave disease?

a. Increased total and free T4

b. Suppressed plasma TSH

c. Exophthalmos

d. Decreased thyroid radioactive iodine uptake

6. Patients with Graves' disease have high circulating levels of thyroid hormones. Graves' disease is characterized by the following, except?

a. Excess release of TSH from the anterior pituitary

b. Hyperactivation of TSH receptor in thyroid glands

c. Presence of TSH receptor antibodies causing receptor activation

d. Decreased TSH levels

7. Which of the following is not the metabolic function of thyroid hormones?
   a. Glycogenolysis
   b. Gluconeogenesis
   c. Glucose oxidation
   d. Glycogenesis

Answer

1.	d	2.	d	3.	a
4.	c	5.	d	6.	a
7.	d				

CHAPTER

36

Sthoulya - Karshya

स्थौल्य (Sthaulya)

Introduction

Excessive accumulation of fat in the region of breast, hip, belly etc. of the body leading to various complications is identified as *Medovṛddhi* or *Medo-Roga*. The patient of *Medo-Vṛddhi* faces such indispositions as excessive sweating, hunger and thirst, he emits bad smell, feels weakness and difficulty in sexual intercourse and in movement. The indisposition may lead to many complications e. g. diabetes, heart disease, hemiplegia and arthritis.

स्थौल्य हेतु एवं संप्राप्ति (Sthaulya Hetu evam Samprāpti)

रसनिमित्तमेव स्थौल्यं कार्श्यं च। तत्र श्लेष्मलाहारसेविनोऽध्यशनशीलस्याव्यायामिनो दिवास्वप्नरतस्य चाम एवान्नरसो मधुरतरश्च शरीरमनुक्रामन्नतिस्नेहान्मेदो जनयति। तदतिस्थौल्यमापादयति। तमतिस्थूलं क्षुद्रश्वासपिपासाक्षुत्स्वप्नस्वेदगात्रदौर्गन्ध्यक्रथनगात्रसादगद्गदत्वानि क्षिप्रमेवाविशन्ति, सौकुमार्यान्मेदसः सर्वक्रियास्वसमर्थः; कफमेदोनिरुद्धमार्गत्वाच्चाल्पव्यवायो भवति आवृतमार्गत्वादेव शेषा धातवो नाप्यायन्तेऽत्यर्थमतोऽल्पप्राणो भवति, प्रमेहपिडकाज्वरभगन्दरविद्रधिवातविकाराणामन्यतमं प्राप्य पञ्चत्वमुपयाति। सर्व एव चास्य रोगा बलवन्तो भवन्त्यावृतमार्गत्वात् स्रोतसाम्, अतस्तस्योत्पत्तिहेतुं परिहरेत्॥

S.S.Su. 15/32

मेदस्तु सर्वभूतानामुदरेष्वस्थिषु स्थितम्।
अत एवोदरे वृद्धिः प्रायो मेदस्विनो भवेत्॥
मेदसाऽऽवृतमार्गत्वाद्वायुः कोष्ठे विशेषतः।
चरन् सन्धुक्षयत्यग्निमाहारं शोषयत्यपि॥
तस्मात् स शीघ्रं जरयत्याहारमभिकाङ्क्षति।
विकारांश्चाप्नुते घोरान् कांश्चित् कालव्यतिक्रमात्॥
एतावुपद्रवकरौ विशेषादग्निमारुतौ।
एतौ तु दहतः स्थूलं वनदावो वनं यथा॥
मेदस्यतीव संवृद्धे सहसैवानिलादयः।
विकारान् दारुणान् कृत्वा नाशयन्त्याशु जीवितम्॥

M.Ni. 34/4-8

The etiological factors of the *Sthaulya* are as follows : Excessive eating, intake of heavy, cold, sweet, fatty food substances, *Kapha Vardhaka* foods & drinks and activities; lack of physical exercises, day sleep, lack of sexual intercourse and mental exercise, cheerfulness, intake of sweet substances by mother during pregnancy, defective genes & other *Dhātu* saturating measures.

स्थौल्य संप्राप्ति (Sthaulya Samprāpti)

Pathogenesis of *Sthaulya* described below : Due to etiological factors *Kapha Doṣa* become aggravated & food remains uncooked & turns to more sweet, this *Rasadhātu* circulating throughout the body produces *Medas* because of its unctuousness is known as *Sthaulya.*

Sthaulya person suffers from mild dyspnoea, severe thirst, hunger, sleep, perspiration, bad smell from the body, snoring, debility of the body & stammering.

Due to softness of *Meda* the person becomes incapable of all activities; because of obstruction in the *Śukravaha Srotas* by *Kapha & Meda* leads to poor sexual pleasure; due to obstruction in other tissue channels the remaining *Dhātus* do not get sufficient nourishment, this leads to lesser strength in all activities. *Sthaulya* persons becomes victims of *Prameha, Prameha Piḍakā, Jvara, Bhagandara, Vidradhi, Vāta Vikara, Śvāsa, Udara roga* & *Urustambha* & such other diseases & death. All the above diseases become very powerful due to obstruction of the *Srotas.*

Abdomen & bones are the chief depositor of *Meda,* the belly gets enlarged in such individuals. The channels of *Vāta* become obstructed by the increased *Meda* inside the abdomen; *Vāta* then begins to act vigorously inside the *Mahāsrotas* as a result *agni* becomes powerful leading to voracious hunger and craves for more quantity of food. In due course of time more distressing conditions prevails. Combination of both *Agni* & *Vāta* are more harmful just as forest fire destroys the forest, these destroy the body. Due to enhancement of *Meda* all the three *Doṣās* become aggravated & causes many serious complications including death.

Pathogenesis of Sthaulya due to increased Meda & Kapha According to Suśruta

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphādi Doṣās*
↓
Disturbance of *Agni*
↓
Food remains *Āma* (uncooked) & turns to more sweet (*Madhura Avasthāpaka*)
↓
This *Adhya Rasadhātu* circulating throughout the body produces *Medas* because of its unctuousness
↓
Sthaulya.

Pathogenesis of Sthaulya due to increased Meda & Kapha Associated with Powerful Agni & Vāta

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphādi Doṣās*
↓
Increase in quantity of *Meda* due to similar properties with *Kapha*
↓
Abdomen & bones are the chief depositor of *Meda* (the belly gets enlarged in such individuals)
↓
The channels of *Vāta* become obstructed by the increased *Meda* inside the abdomen
↓
Vāta then begins to act vigorously inside the *Mahāsrotas* as a result *agni* becomes powerful leading to voracious hunger and craves for more quantity of food
↓
Eat more quantity of food
↓
Sthaulya.
↓
In due course of time more distressing conditions prevails
↓
Combination of both *Agni* & *Vāta* are more harmful just as forest fire destroys the forest, these destroy the body
↓
Due to enhancement of *Meda* all the three *Doṣās* become aggravated & causes many serious complications and diseases
↓
Prameha, Prameha Piḍakā, Jvara, Bhagandara, Vidradhi, Vāta Vikara, Śvāsa, Udara roga & *Urustambha* & such other diseases & death.
(These diseases become very powerful due to obstruction of the *Srotas*)

स्थौल्य लक्षण (Sthaulya Lakṣaṇa)

मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः ।
अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते ॥
M.Ni. 34/9

Person can be diagnosed as *Sthaulya* when his buttocks, abdomen & breasts begin to show movement due to excess accumulation of *Meda* in these areas. Contrary to his age there is a disproportionate in relation to build & enthusiasm (B.P.M. 39/9-10).

Kuṣtha, Visarpa, Bhagandara, Jvara, Atisāra, Prameha, Arśa, Ślīpada, Apaci, Kāmalā may develop in *Sthaulya* persons. Due to the bad smell of sweat minute worms may develop in the skin (B.P.M. 39/9-10).

मेदस्यतीव संवृद्धे सहसैवानिलादयः ।
विकारान्दारुणान्कृत्वा नाशयन्त्याशु जीवितम् ॥
मेदोमांसातिवृद्धत्वाच्चलस्फिगुदरस्तनः ।
अयथोपचयोत्साहो नरोऽतिस्थूल उच्यते ॥
BPM. 39/8-9

When *Meda Dhātu* becomes greatly increased, then *Vātādi Doṣās* give rise to many dreadful diseases & destroy the life of the individuals.

Due to excess enhancement of *Meda* & *Māmsa* leading to pendulous movement of buttocks, abdomen & breast, disproportionate increase of body parts & lack of enthusiasm to work is known as *Atisthūla*.

उपशय/पथ्य (Upaśaya/Pathya)

Persons indulging in physical exercise, worry, sexual intercourse, long walk, intake of honey, remaining awake at night and taking *Yava* and *Śyāmaka* as food gets rid of obesity. (B.R. 39/1)

If the patient wants to be free from obesity, then he should gradually increase *Asvapna* (remaining awake at night), sexual intercourse, physical exercise and mental activities. (B.R. 39/2)

The following measures and eatables are very much helpful in curing the problem of obesity: sorrow, labour, waking during night, enjoying sex, unguents, *Laṅgana* (fasting), facing the sun, riding elephant and horse, traveling, *Apatarpaṇa*, *Purātana*, *Vaiṇava*, *Koradūṣa*, *Kodarva*, *Śyāmāka*, *Nīvāra*, *Priyaṅgu*, barley (*Yava*), *Kulattha*, gram (*Caṇaka*), lentil, *Mudga*, pigeon peas, honey, poppy of paddy, edibles and juices having bitter tastes, buttermilk, liquor, *Ciṅgata* variety of fishes, fried brinjal, *Tṛiphalā*, *Guggulu*, *Lauha Bhasma*, *Tṛikaṭu*, mustard oil, cardamom, all dry materials, sesame oil, leafy vegetables, poultice of *Agara*, warm water, *Śilājatu*, drinking water before meals. (B.R. 39/67-71)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures and diets are considered to be harmful for the patients who are suffering from obesity: bathing with cold water, *Rasāyana*, fresh *Śāli* type of rice, wheat, such activities as lying or resting on luxury cushions, products of milk, products of sugarcane-jaggery, etc., fried nuggets prepared out of the *Urada* beans, taking huge amount of meals, *Snehana Karma* (massage), eating fish and meat, sleeping during daytime, sporting garlands and perfumed oil and sandalwood, eating sweet products, drinking lot of water after taking meals and *Vamana* therapy. (B.R. 39/72-74).

कार्श्य (Kārśya)

निरुक्ति एवं परिभाषा (Nirūkti and Paribhāṣa)

The term *'Kārśya'* is derived from the word *'Kṛśa'* meaning *'Tanūkaraṇe'* which means making thin or emaciated. Hence, the word *'Kārśya'* means emaciation of the body.

कृशस्य भावः कार्श्यः । *Śabdakalpdruma*

A factor which expresses the thinness or leanness or emaciation of the body is known as *Kārśya*.

कार्श्यं कृशकरणम् । *Śabdakalpdruma*

Skinniness or slenderness or emaciation of the body is called *Kārśya.*

अपचयः कार्श्यः। *S.S.Su. 10/5 (Dalhaṇa)*

Diminution or decrease or degradation of the body is called *Kārśya.*

कार्श्यं मांसक्षयः ॥ *S.S.Su. 15/3 (Dalhaṇa)*

Diminution of muscle tissue is called *Kārśya.*

कार्श्यमिति कृशता शरीरस्य वातप्रकृतिजत्वात् ॥
Āḍd.Śa.Prā. 15/3

Emaciation of the body due to predominance of *Vāta* is called *Kārśya.*

शुष्कस्फिगुदरग्रीवो धमनीजालसन्ततः ।
त्वगस्थिशेषोऽतिकृशः स्थूलपर्वा नरो मतः ॥
C.S.Su. 21/15

Atikṛśa or *Kārśya is* characterised by emaciation of buttocks, abdomen and neck, prominence of network of vessels all over the body associated with leaness and bony prominence with large joints.

सम्प्राप्ति घटक (Saṁprāpti Ghaṭaka)

Doṣa : *Vāta pradhāna tridoṣa*
Dūṣya : *Rasa*
Agni : *Jaṭharāgni māndhya*
Āma : *Jaṭharāgni māndhyajanya Āma*
Srotas : *Rasa, Māṁsa* and *Medovaha*
Srotoduṣṭi : *Saṅga*

Sañcāra sthāna: *Sarva Rasāyanīs*
Adhiṣṭhāna : *Sarva Sarīra*
Udbhava sthāna: *Āmapakvāśaya*
Vyakta sthāna : *Sarvāṅga* especially *Sphik, Udara* and *Grīva*
Rogamārga : Bāhya

कार्श्य निदान (Kārśya Nidāna)

तत्र पुनर्वातलाहारसेविनोऽतिव्यायामव्यवायाध्ययनभयशोकध्यानरात्रिजागरणपिपाशाक्षुत्कषायाल्पाशनप्रभृतिभिरुपशोषितो रसधातुः शरीरमननुक्रामन्नल्पत्वान्न प्रीणाति, तस्मादतिकार्श्यं भवति, सोऽतिकृशः क्षुत्पिपासाशीतोष्णवातवर्षभारादानेष्वसहिष्णुर्वातरोगप्रायोऽल्पप्राणश्च क्रियासु भवति, श्वासकासशोषप्लीहोदराग्निसादगुल्मरक्तपित्तानामन्यतममासाद्य मरणमुपयाति । सर्व एव चास्य रोगा बलवन्तो भवन्त्यल्पप्राणत्वात् । अतस्तस्योत्पत्तिहेतुं परिहरेत् ॥ *S.S.Su. 15/33*

Due to indulgence of food which increase *Vāta* along with excessive physical exercises, sexual intercourse, study, fear, grief, worry, keeping awake at nights, suppression of thirst & hunger, consumption of astringent taste food, less intake of food etc. leading to less formation & dryness of *Rasadhātu*. The same *Rasadhātu* circulate all over the body fails to nourish the other *Dhātus* because of its inadequate quantity. This is known as *Kārśya*.

Kārśya people becomes incapable of tolerating hunger, thirst, cold, heat, breeze, rain, carrying heavy loads, *Vātaja Vikara* & less physical activities.

Diseases manifest quickly in *Kārśya* people are as follows : *Śvāsa, Kāsa, Śoṣa, Plīha, Udara, Agnisāda, Gulma, Raktapitta, Arśa, Grahaṇi* & lastly death. All the above diseases are serious because of less strength of the body.

कार्श्य लक्षण (Kārśya Lakṣaṇa)

शुष्कस्फिगुदरग्रीवाधमनीजालसन्ततिः ।
त्वगस्थिशोषोऽतिकृशः स्थूलपर्वाननो मतः ॥
BPM 40/3

Emaciation, wasting of the buttocks, abdomen & neck, appearance of network of veins & tendons all over the body, prominence of skin, bones & joints.

Please refer first volume of the author for more information about *Kārśya*. Don't diagnose *Sthaulya* & *Kārśya* by seeing ones own body.

कश्चिदन्यः कृशोऽतीव बलवान्दृश्यते तदा ॥
आधानसमये यस्य शुक्रभागोऽधिको भवेत् ।
मेदोभागस्तु हीनः स्यात्स कृशोऽपि महाबलः ॥
भेदसस्त्वधिकोयस्य शुक्रभागोऽल्पको भवेत् ।
स स्निग्धोऽपि सुपुष्टोऽपि बलहीनो विलोक्यते ॥
BPM 40/6

An emaciated person who at the time of his birth had more portion of *Śukrabhāga* & less amount of *Medobhāga* will be very strong though appears as emaciated.

On the contrary one who at the time of his birth had more portion of *Medobhāga* & less portion of *Śukrabhāga* will be poor in strength though those persons appear as were nourished & unctous.

साध्यासाध्यता (Sādhyāsādhyata)

कार्श्यमेव वरं स्थौल्यात् न हि स्थूलस्य भेषजम् ।
बृंहणं लङ्घनं वाऽलमतिमेदोग्निवातजित् ॥
A.H.Sū. 14/31

स्थैल्यात् कायर्श्स्य सुखसाध्यत्वमाह ।
A.H.Sū. 14/31(Indu)

Reasonably *Kārśya* is easily curable than *Sthaulya,* because there is no treatment for *Sthaulya;* neither *Bṛṁhaṇa* nor *Laṅghana* are enough to control *Atimeda* (Excessive fat), *Agni* (Digestive fire) and *Vāta.*

स्वभावादतिकार्श्यो यः स्वभावादल्पपावकः । स्वभावादबलो यश्च-तस्य नास्ति चिकित्सितम् ॥ (B.P.M. 40/11)

There is no treatment to *Kārśya* if it occurs due to *Svābhāva* (Nature or birth), *Svabhāvādalpapāvakaḥ* (Whose digestive fire is weak) and *Svabhāvādabalo* (Whose strength is low).

Under-nutrition

Starvation and famine

1. There remain regions of the world, particularly rural Africa, where under-nutrition due to famine is endemic, the prevalence of BMI < 18.5kg/mg in adults is as high as 20%, and

growth retardation due to under nutrition affects 50% of children.

2. WHO reports that under-nutrition is responsible for more than half of all childhood deaths world-wide.
3. Starvation is manifest as marasmas (malnutrition with marked muscle-wasting); or kwashiorkor (malnutrition with oedema).
4. Growth retardation is due to deficiency of key nutrients, e.g. protein, zinc, potassium, phosphorus and sulphur.

Classification of under-nutrition in adults by body mass index (weight/height2)

BMI (kg/m^2	Classification
> 20	Adequate nutrition
18.5-20	Marginal
< 18.5	Under-nutrition
17-18.4	Mild
16-17	Moderate
< 16	Severe

Causes of under-nutrition and weight loss in adults

Decreased energy intake

- Famine
- Persistent regurgitation or vomiting
- Anorexia, including anorexia nervosa
- Malabsorption (e.g. small intestinal disease)
- Maldigestion (e.g. pancreatic exocrine insufficiency) I

Increased energy expenditure

- Increased BMR (thyrotoxicosis, trauma, fever, cancer cachexia)
- Excessive physical activity (e.g. marathon runners)
- Energy loss (e.g. glycosuria in diabetes)
- Impaired energy storage (e.g. Addison's disease, phaeochromocytoma)

Clinical assessment

1. In starvation, the severity of malnutrition can be assessed by anthropometric measurements as mentioned above.
2. Demispan and mid-arm circumference measurements are most useful in monitoring progress during treatment.

The clinical features of severe under-nutrition in adults include:

- Weight loss
- Thirst, craving for food, weakness feeling cold
- Nocturia, amenorrhoea impotence
- Lax, pale, dry skin with loss of turgor and, occasionally, pigmented patches
- Cold and cyanosed extremities,

Pressure sores

- Hair thinning or loss (except in adolescents)
- Muscle-wasting, best demonstrated by the loss of the temporalis and periscapular muscle and reduced mid-arm circumference
- Loss of subcutaneous fat, reflected in reduced skin fold thickness and mid-arm circumference
- Hypothermia, bradycardia, hypotension small heart
- Oedema, which may be present without hypoaulbuminaemia ('famine oedema')
- Distended abdomen with diarrhoea
- Diminished tendon jerks
- Apathy, loss of initiative, depression, introversion, aggression if food is near by
- Susceptibility to infections

Consequences of Under nutrition

1. Under-nutrition often leads to vitamin deficiencies, especially of thiamin, folate and vitamin C
2. Diarrhoea can lead to depletion of sodium, potassium and magnesium.
3. The high mortality rate in famine situations is often due to outbreak of infection, e.g. typhus or cholera, but the usual signs of infection may not be apparent.
4. In advanced starvation, patients become completely inactive and may assume a flexed, fetal position.
5. In the last stage of starvation, death comes quietly and often quite suddenly.
6. The very old are most vulnerable.

7. All organs are atrophied at necropsy, except the brain, which tends to maintain its weight.

Investigations

1. Plasma free fatty acids are increased and there is ketosis and a mild metabolic acidosis.
2. Plasma glucose is low but albumin concentration is often maintained berceuse the liver still functions normally.
3. Insulin secretion is diminished, glucagon ond cortisol tend to increase.
4. Reverse T_3, replaces normal triiodothyronine.
5. The resting metabolic rate falls, partly because of reduced lean body mass and partly because of hypothalamic compensation .
6. The urine has a fixed specific gravity and creatinine excretion becomes low.
7. There may be mild anaemia, leucopenia and thrombocytopenia.
8. The erythrocyte sedimentation rate is normal unless there is infection.
9. The electrocardiogram shows sinus bradycardia and low voltage.

Differential diagnosis of Kārśya

S.No.	*Kārśya*	*Dhātukṣaya*
Type of *Vyādhi*	*Apatarpaṇajanya Vyādhi* & *Kṛṣa pramehi*	*Apatarpaṇajanya Vyādhi*
Etiology	Excessive consumption of rough foods and drinks • Fasting • Less intake of food • Excessive exposure to purificatory therapies • Grief • Suppression of natural urges including the urge for sleep • Distrubed sleep • Application of non unctuous substances in excess • Excessive indulgence in bath • By birth due to genes • Old age • Suffering from chronic disorder	The following diets are responsible for the depletion of dhatus c S Ci 1-2/3 *Grāmya Āhara* (domestic food and lifestyle) *Grāmya Āhara* (poor quality diet) is described below: • Excess intake of *Amla* (Sour), *Lavaṇa* (salt), *Kaṭu* (pungent) taste food • Excess intake of *Kṣāra* (alkaline), *Suṣka Sāka* (dry green vegetables), *Suṣka Māmsa* (dry meat), *Tila/ Acurnata Tila* (seasum seeds), Tila *Palala/ Tila Curṇa* (paste of sesame seeds and pastries) • *Pistanna bhojana*- food prepared from flour like cake and pastries • *Viruddha Nava Suka Sami Dhānya* (germinated cereals and pulses, freshly harvested corns with bristles and pulses) • *Viruddha* (intake of antagonistic food) • *Asāthmya* (unwholesome foods) • *Klinna, Guru, Puti Paryusita Bhojana* (softened, moist, heavy, putrid and stale food), • *Viṣamāśana* (eating at irregular intervals) • *Adhyāśana* (eating too soon after a meal without the digestion of previous meal) **Habits that cause illness -** • *Divāsvapna* (day sleep) • *Stree Madya Nitya* (habitual indulgence in sex and alcohol) • *Visama* and *Ati Vyāyama* (irregular and excessive exercise),

		• *Bhaya* (fear), *Krodha* (anger), *Śoka* (grief), *Lobha* (greed), *Moha* (confusion), *Ayasa Bahula* (exhaustion due to overstrain).
Does not tolerate	***Atikṛśa* person does not tolerate** व्यायाममतिसौहित्यं क्षुत्पिपासामयौषधम् । कृशो न सहते तद्वदतिशीतोष्णमैथुनम् । *C.Su 21/13* • Physical exercise • Over saturation or excess consumption of food • Hunger, thirst • Diseases • Moderate and powerful drugs • Excessive cold and heat • Sexual intercourse Fail to perform normal physiological daily routines	
Diseases/effects	**The following diseases manifest in *atikṛśa* individual** प्लीहा कासः क्षयः श्वासो गुल्मोऽर्शांस्युदराणि च । कृशं प्रायोऽभिधावन्ति रोगाश्च ग्रहणीगताः । *C.Su 21/14* • *Plīhā roga* • *Kāsa* • *Kṣaya* • *Śvāsa* • *Gulma* • *Arśa* • *Udara roga* • *Grahaṇi doṣa*	**Effect of *Grāmya Āhara Sevana* -** The intake of such *Grāmyāhara* (domestic food and Lifestyle) causes 1. *Sithilata*- slackening of muscle tissue compactness 2. *Vimucyante Sandhi* - Looseness/detachment of joint 3. *Vidahyate Rakta* - Blood gets inflamed & burnt 4. *Viṣyandate Alpa Meda* - increased free fatty acids 5. *Na Sandhiyate Asthiṣu Majja*- Marrow does not mature in bones 6. *Śukra Na Pravartate*-less formation of Semen 7. *Kṣaya Ojas*- Oja deteriorates
Clinical presentations	**Prominent features of *atikṛśa* individual are :** शुष्कस्फिगुदरग्रीवो धमनीजालसन्ततः । त्वगस्थिशेषोऽतिकृशः स्थूलपर्वा नरो मतः। *C.Su 21/15* • Wasting prominently marked in buttocks, abdomen and neck • Prominent vascular net work in the body • Remnants of skin and bone • Joints are prominently seen	The person suffers from malaise, despair, hypersomnia and excessive sleepiness, drowsiness, lassitude, lack of enthusiasm, difficulty in breathing, unable to execute physical and mental activities, loss of memory, intellect and luster, becomes resort of illness and thus does not enjoy the normal life span
Pathogenesis	Develop due to Vatakopanidaṇa which result in less formation and dryness of *Rasa dhātu* followed by deprivation of subsequent *dhātus*	Anuloma or pratiloma type of depletion of *Dhātus*

Weight Loss

Ask the Following Questions:

Is there fever? The presence of fever would suggest an infectious disease, such as tuberculosis, AIDS, brucellosis, and typhoid fever, but collagen diseases and neoplasms should not be forgotten.

Is there anorexia? The presence of anorexia may be related to a febrile process, but if there is no fever one should consider the possibility of Addison's disease, anorexia nervosa, Simmonds'disease, drug abuse, poisoning, such as arsenic poisoning, scurvy, malabsorption syndrome, uremia, and liver failure. There may also be a neoplasm.

Is there lymphadenopathy? The presence of generalized lymphadenopathy should suggest leukemia, sarcoidosis, and lymphoma, as well as infectious disease processes.

Is there an abdominal mass? An abdominal mass may be an enlarged spleen, a pancreatic carcinoma, an enlarged liver, or renal mass. These masses would suggest disease of those organs. The mass also may be a carcinoma of the stomach or intestine.

Is there hyperpigmentation? The presence of hyperpigmentation would suggest Addison's disease.

Is the appetite normal or increased? The presence of a normal or increased appetite in the presence of weight loss should suggest hyperthyroidism and diabetes mellitus. The patient also may be taking thyroid hormone medication in increased quantities.

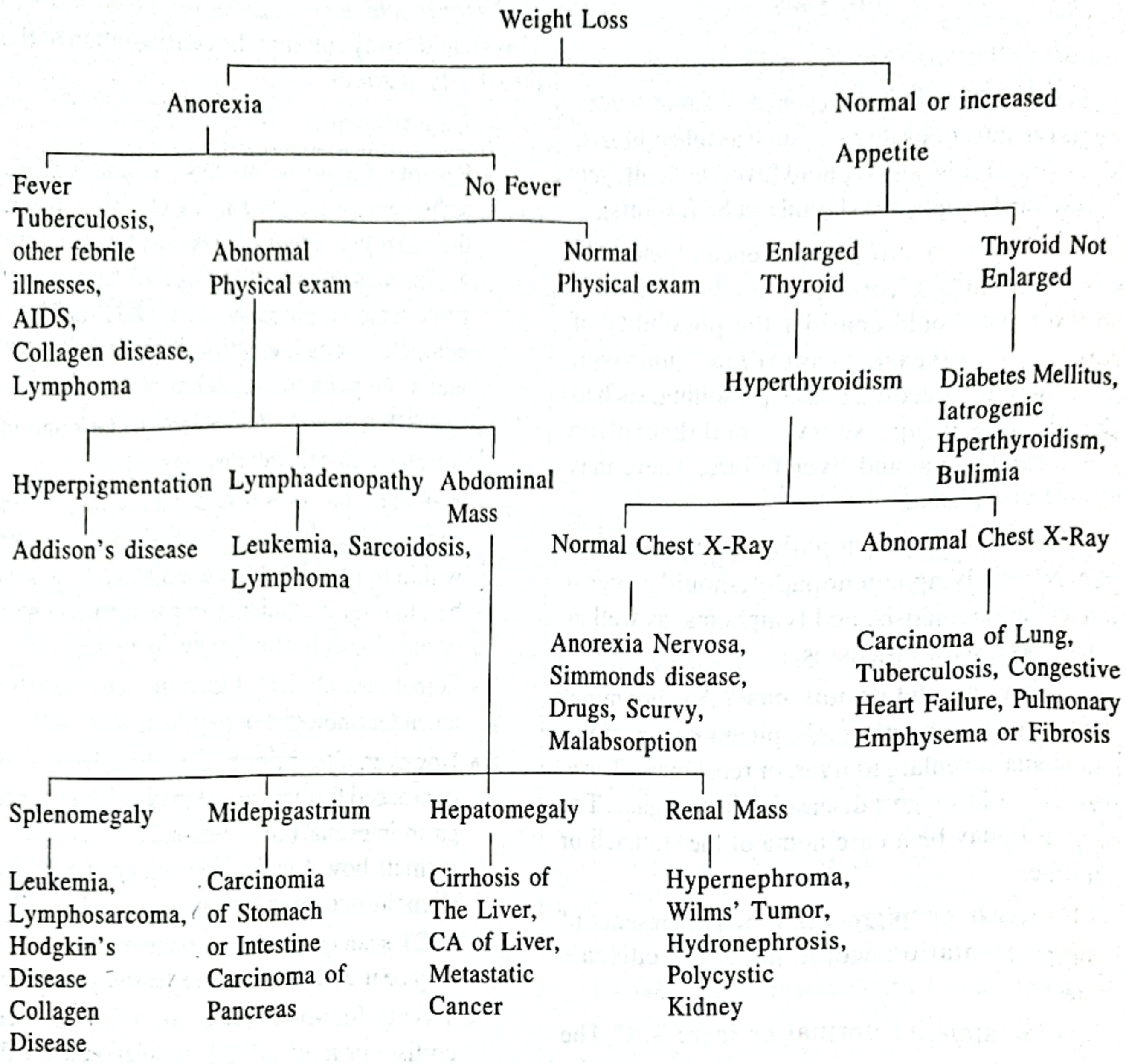
Is the thyroid gland enlarged? The presence of an enlarged thyroid would suggest hyperthyroidism. One should also look for a focal thyroid mass which might be a toxic adenoma.

Is the chest x-ray abnormal? Abnormalities found on x-ray that may induce weight loss are carcinoma of the lung, tuberculosis, congestive heart failure, pulmonary emphysema, and fibrosis.

Is there a history of risky sexual behavior? This should always prompt the consideration of the possibility of AIDS.

Diagnostic Workup

- Routine diagnostic studies include a CBC, sedimentation rate, urinalysis, chemistry panel, thyroid panel, serum amylase and lipase, febrile agglutinins, tuberculin test, ANA titer, serum protein electrophoresis, serum B12 and folic acid, chest x-ray, electrocardiogram (EKG), and a flat plate of the abdomen.
- An HIV antibody titer needs to be done in selected clinical circumstances.
- A stool for fat, trypsin, occult blood, and ovum and parasites should be done. If these tests are within normal limits or are unrevealing, it is best to refer the patient to a gastroenterologist or oncologist for further evaluation.
- Sometimes, clinical clues suggest the need for an endocrinologist or psychiatrist as well.
- However, if the primary care physician wishes to proceed further, he/she may order an upper gastrointestinal (GI) series and esophagogram, a small bowel series, barium enema, and a sigmoidoscopic examination.
- A CT scan of the chest, abdomen, and pelvis may be useful, but it is an expensive procedure.
- Twenty-four-hour urine collection for free cortisol or rapid ACTH stimulation test will diagnose Addison's disease and hypopituitarism.
- Quantitative stool fat and D-xylose absorption or a simple glucose tolerance test will diagnose some cases of malabsorption syndrome.
- Endoscopic procedures, including laparoscopy, and even an exploratory laparotomy, have their place in the diagnostic workup. However, it is always best to enlist the help of specialists before considering these procedures, even if one is located in an isolated community. If a trial of a nutritional supplement halts the weight loss, depression is most likely the cause.


Weight Loss
Anorexia
Normal or increased Appetite
Fever
Tuberculosis, other febrile illnesses, AIDS, Collagen disease, Lymphoma
No Fever
Abnormal Physical exam
Normal Physical exam
Enlarged Thyroid
Thyroid Not Enlarged
Hyperthyroidism
Diabetes Mellitus, Iatrogenic Hperthyroidism, Bulimia
Hyperpigmentation
Lymphadenopathy
Abdominal Mass
Addison's disease
Leukemia, Sarcoidosis, Lymphoma
Normal Chest X-Ray
Abnormal Chest X-Ray
Anorexia Nervosa, Simmonds disease, Drugs, Scurvy, Malabsorption
Carcinoma of Lung, Tuberculosis, Congestive Heart Failure, Pulmonary Emphysema or Fibrosis
Splenomegaly
Midepigastrium
Hepatomegaly
Renal Mass
Leukemia, Lymphosarcoma, Hodgkin's Disease Collagen Disease
Carcinomia of Stomach or Intestine Carcinoma of Pancreas
Cirrhosis of The Liver, CA of Liver, Metastatic Cancer
Hypernephroma, Wilms' Tumor, Hydronephrosis, Polycystic Kidney

	Fever	Appetite	Abdominal Mass	Polyuria	Lymphadenopathy
Infectious disease	Yes	Decreased	Infrequet	No	Yes
Neoplasm	No	Decreased	Often	No	Maybe
Lukemia	Occasional	Decreased	Maybe	No	Yes
Hyperthyroidism	No	Increased	No	Yes	No
Diabetes mellitus	No	Increased	No	Yes	No
Anorexia nervosa	No	Decreased	No	No	No
Bulimia	No	Normal	No	No	No

MCQs

1. आहारपरिणामान्तेभूयश्चलभतेबलम is clinical feature of
   a. व्यपेताहिक्का (Vyapeta Hikk
1. "*Medasavritamargatvadvayuhkoshtevisheshtah*" explained in context to
   a. *Atisthoola* b. *Medasavritavata*
   c. *Atisara* d. *Udararoga*
2. *Dhatu* responsible for *Karsya*
   a. *Medas* b. *Mamsa*
   c. *Rasa* d. *Sukra*
3. तस्यह्यतिमात्रमेदस्विनोमेदएवोपचीयतेनतथेतरेधातवः_____
   a. *Krichravyavayata* b. *Ayushohrasa*
   c. *Dourbalya* d. *Javoparodha*
4. शैथिल्यात्सौकुमार्याद्गुरुत्वाच्चमेदसो_____
   a. *Dourbalya* b. *Javoparodha*
   c. *Krichravyavayata* d. *Ayushohrasa*
5. *Asamatwa* of *Dhatus* in *Sthula* result in
   a. *Dourbalya* b. *Javoparodha*
   c. *Dourbalya* d. *Krichravyavayata*

Answer

1.	a	2.	c	3.	b
4.	b	5.	c		

CHAPTER
37

Obesity

1. Obesity is widely regarded as a pandemic, with potentially disastrous consequences for human health.
2. In developing countries, average national rates of obesity are low, but these figures may disguise: high rates of obesity in urban communities; for example, nearly one-quarter of women in urban India are overweight.
3. There is increasing public awareness of the health implications of obesity.
4. Many patients will seek medical help for their obesity, others will present with one of the complications of obesity, and increasing numbers are being identified during health screening examinations.
5. It is suggested that obesity at age 40 years can reduce life expectancy by up to 7 years for non-smokers and by 13 years for smokers.
6. Coronary heart disease is the major cause of death but cancer rates are also increased in the overweight, especially colorectal cancer in males and cancer of the gallbladder, billiary tract, breast, endometrial and cervix in females.
7. Obesity has little effect on life expectancy at > 70 years, but the obese do spend a greater proportion of their active life disabled.
8. Epidemic obesity has been accompanied by epidemic of type 2 diabetes and osteoarthritis, particularly of the knee.
9. Obesity may have profound psychological consequences, compounded by stigmatisation of the obese in many societies.

Complications of obesity

Obesity has adverse effects on both mortality and morbidity :

Risk factors	Outcomes
1. Metabolic syndrome Type 2 diabetes Hypertension Hyperlipidaemia	Coronary heart disease Stroke Diabetes complications
2. Liver fat accumulation Cirrhosis	Non-alcoholic steatohepatitis
3. Restricted ventilation Sleep apnoea Respiratory failure (Pickwickian syndrome)	Exertional dyspnoea
4. Mechanical effects of weight	Urinary incontinence Osteoarthritis Varicose veins
5. Increased peripheral steroid interconversion in adipose tissue	Hormone-dependent cancers (breast, uterus) Polycystic ovary syndrome (infertility, hirsutism)
6. Others	Psychological morbidity (low self-esteem, depression) Socioeconomic disadvantage (lower income, less likely to be promoted) Gallstones Colorectal cancer Skin infections (groin and sub mammary candidiasis; hidradenitis)

Body fat distribution

1. Increased intra-abdominal fat causes central ('abdominal', visceral', 'android' or'appleshaped') obesity and it is more common in men and is more closely associated with type 2 diabetes, the metabolic syndrome and cardiovascular disease.
2. Increased subcutaneous fat accumulation causing 'generalised' ('gynoid' or 'pearshaped') obesity

Aetiology

Accumulation of fat results from a discrepancy between energy consumption and energy expenditure which is too large to be defended by the hypothalamic regulation of BMR.

Some reasons for the increasing prevalence of obesity-the 'obesogenic' environment

1. Increasing energy intake
   - Portion sizes
   - Energy-dense food
   - Snacking and loss of (mainly fat) regular meals
   - ↓ Affluence
2. Decreasing energy expenditure
   - Car ownership
   - ↓ Sports in schools
   - ↓ Walking to school/work
   - Time spent on computer games and watching TV
   - ↓ Manual labour
   - Automation;
   - Central heating

Potentially reversible causes of weight gain :

1. Endocrine factors
   - Hypothyroidism
   - Hypothalamic tumours or injury
   - Cushing's syndrome
   - Insulinoma
2. Drug treatments
   - Tricyclic antidepressants
   - Corticosteroids
   - Sulphonylureas
   - Sodium valproate
   - Oestrogen-containing
   - Contraceptive pill

Susceptibility to obesity

1. Susceptibility to obesity and its adverse consciences undoubtedly varies between individuals.
2. It is not true that obese subjects have a 'slow metabolism', since their BMR is higher than that of lean subjects.
3. Twin and adoption studies confirm a genetic influence on obesity.

Reversible causes of obesity and weight gain

These patients are distinguished from those with idiopathic obesity by their short history, with a recent marked change in the trajectory of their adult weight

Clinical assessment and investigations

1. Severity of obesity can be quantified using the BMI
   Quantifying obesity with body mass index (weight/height^2)

Risk of obesity

BMI (kg/m2)	Classification*	Comorbidity
18.5-24.9	Normal range	Negligible
25.0-29.9	Overweight	Mildly increased
> 30.0	Obese	
30.0-34.9	Class I	Moderate
35.0-39.9	Class II	Severe
> 40.0	Class III	Very severe

2. A waist circumference of > 102cm in men or > 88cm in women indicates that the risk of metabolic and cardiovascular complications of obesity is high.
3. A dietary history may be helpful in guiding dietary advice, but is notoriously susceptible to under-reporting of food consumption.
4. It is important to consider 'pathological eating behaviour (such as binge eating, nocturnal eating or bulimia, which may be the most important issue to address in some patients.
5. Alcohol is an important source of energy intake and should be considered in detail.
6. The history of weight gain may help diagnose underlying causes. such as hypothyroidism or Cushing's syndrome.
7. Obese patients should have thyroid function tests performed on one occasion
8. An overnight dexamethasone suppression test or 24-hour urine free cortisol if syndrome is suspected.

9. Monogenic and 'syndromic' causes of obesity are usually only relevant in children presenting with severe obesity.
10. The impact of obesity on the patient's life and work is a major consideration. Assessment of other cardiovascular risk factor is important.
11. Blood pressure should be measured with a large cuff, if required.
12. blood glucose and a serum lipid profile
13. Elevated serum transaminases occur in patients with non-alcoholic fatty liver disease.

Differential diagnosis of obesity

- **Is there associated hyperphagia :** If the patient recognizes that he or she has a ravenous appetite or eats more than is necessary, the possibility of an insulinoma or Frohlich's syndrome should be considered.
- **Is the obesity centripetal:** The presence of centripetal obesity, especially with moon facies, should suggest Cushing's syndrome.
- Is the obesity mainly of the lower extremities: This finding would suggest lipodystrophy.
- **Is there mental retardation:** The presence of mental retardation should suggest LaurenceMoonBardetBiedl syndrome.
- **What is the sex of the patient:** In male patients, one should consider Klinefelter's syndrome, and in female patients, one should consider polycystic ovary.
- **What drugs is the patient on :** Many drugs may cause obesity, most notably the tricyclic antidepressants and corticosteroids.

Diagnostic Workup

- Routine laboratory tests include a CBC, urinalysis, chemistry panel, 2-hour postprandial blood sugar, and thyroid profile.
- If an insulinoma is strongly suspected, plasma insulin, C-peptide, a 24- to 36-hour fast, a 5-hour glucose tolerance test, and tolbutamide tolerance test may be done.
- If Cushing's syndrome is suspected, a 24-hour urine free cortisol and cortisol suppression test should be done.
- Pelvic ultrasound will help diagnose polycystic ovaries.
- Chromosomal analysis will help diagnose Klinefelter's syndrome.
- Perhaps a psychiatrist should be consulted.

MCQs

1. The formula for the body mass index is ______.
   a. A person's weight in kilograms squared divided by the square of his/her height in meters (kg^2/m^2)
   b. A person's weight in kilograms squared divided by his/her height in meters (kg^2/m)
   c. A person's weight in kilograms divided by his/her height in meters (kg/m)
   d. A person's weight in kilograms divided by the square of his/her height in meters (kg/m^2)
2. Which among the following are the causes of weight gain
   a. Cushing's syndrome, Hypothyroidism & Corticosteroids
   b. Hyperthyroidism
   c. Diabetes Mellitus Type 2
   d. Addison's disease
3. Centripetal obesity with moon facies, is seen in
   a. Insulinoma
   b. Cushing's syndrome
   c. Laurence–Moon–Bardet–Biedl syndrome
   d. Hypothyroidism

Answer

1.	d	2.	a	3.	b

CHAPTER

38

Prameha

Introduction

Obstinate urinary disorders including diabetes. It is of twenty types- ten of them are caused by *kapha*, six by *pitta* and four by *vāta*. Urinary disease (Name applied to all urinary diseases, of which there are 21 varieties including diabetes, gonorrhoea).

प्रमेह हेतु (Prameha Hetu)

I. आहारज एवं विहारज कारण (Āhāraja evam Vihāraja Kāraṇa)

आस्यासुखं स्वप्नसुखं दधीनि ग्राम्यौदकानूपरसाः पयांसि ।
नवान्नपानं गुडवैकृतं च प्रमेहहेतुः कफकृच्च सर्वम् ॥

C.Ci. 6/4

दिवास्वप्नाव्यायामालस्यप्रसक्तं शीतस्निग्धमधुरमेद्यद्रवान्नपानसेविनं पुरुषं जानीयात् प्रमेही भविष्यतीति ॥ *S.S.Ni. 6/3*

Etiological factors of Prameha are as follows : Sedentary life style, excessive sleep, curd, meat soup of domestic, aquatic and marshy animlas, milk products, freshly harvested food articles, fresh wine, jaggery preparations and all other *kapha* promoting substances, lazyness, intake of food which are cold, unctuous, sweet, fatty, and liquid.

II. सन्तर्पणोत्थ कारण (Santarpaṇottha Kāraṇa)

All etiological factors mentioned for the manifestation of *Santarpaṇottha Vikara* are also responsible for the development of Prameha (C.S.Su. 23/3-5)

III. आदिबल प्रवृत्त (Ādibala Pravṛtta)

......................प्रभृतिर्गहणान्मेह क्षयादयः ।

S.S.Su. 24/5 (Dalhaṇa)

Prameha manifest due to morbidity of *Śukra* and *Śoṇita*.

IV. तेषां मेदोमूत्रकफावहम् ॥ अन्नपानक्रियाजातं यत्प्रायस्तत्प्रवर्तकम् । *(A.H.Ni. 10/1-2)*

Foods, drinks and activities which aggravates *Meda, Mūtra* and *Kapha* are main etiological factors for the genesis of *Prameha.*

V. द्वौ प्रमेही भवतः-सहजोऽपथ्यनिमित्तश्च । तत्र सहजो मातृपितृबीजदोषकृतः, अहिताहारजोऽपथ्यनिमित्तः । तयोः पूर्वेणोपद्रुतः कृशो रूक्षोऽल्पाशी पिपासुर्भृशं परिसरणशीलश्च भवति; उत्तरेण स्थूलो बह्वाशी स्निग्धः शय्यासनस्वप्नशीलः प्रायेणेति ॥

S.S.Ci. 11/13

It manifests due to two main etiological factors i:e Sahaja (Hereditary/Congenital) and *Apathya nimittaja* (due to incompatible dietetics and activities). It manifests due to defective sperm and ovum of the father and mother respectively.

Apathya nimittaja due to consumption of erratic dietetic pattern and activities.

Prāmeha due to Sahaja etiology is characterised by lean, rough body, eats less amount of food, suffering from severe thirst and moves always.

Apathyanimittaja is characterised by obesity, eats more food, unctuous, always desires to occupy bed, sitting posture and sleep.

प्रमेह संप्राप्ति (Samprāpti of Prameha)

कफः सपित्तः पवनश्च दोषा मेदोऽस्रशुक्राम्बुवसालसीकाः ।
मज्जा रसौजः पिशितं च दूष्याः प्रमेहिणां, विंशतिरेव मेहाः ॥

C.S.Ci. 6/8

वर्णं रसं स्पर्शमथापि गन्धं यथास्वदोषं भजते प्रमेहः ।
श्यावारुणो वातकृतः सशूलो मज्जादिसाद्गुण्यमुपैत्यसाध्यः ॥

C.S.Ci. 6/12

तस्य चैवंप्रवृत्तस्यापरिपक्वा एव वातपित्तश्लेष्माणो यदा मेदसा सहैकत्वमुपेत्य मूत्रवाहिस्रोतांस्यनुसृत्याधो गत्वा बस्तेर्मुखमाश्रित्य निर्भिद्यन्ते तदा प्रमेहान् जनयन्ति ॥

S.S.Ni. 6/4

Improperly processed *Vāta, Pitta* and *Kapha* mixes with *Dūṣyās i:e Medas, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa* in the *Mūtravaha Srotas* leading to manifestation of twenty types of *Prameha.*

20 types of *Pramehās* acquires the colour, taste, touch and smell according to the respective *Doṣa.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi Doṣās*
↓
vitiate *Dūṣyās i:e Medas, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa*
↓
Get localised in the *Mūtravaha Srotas*
↓
Prameha
↓
Negligence of *Prameha* Leads to *Madhumeha*

प्रमेह संप्राप्ति घटक (Samprāpti Ghaṭaka of Prameha)

Components of *Samprāpti* are as follows:-

दृष्ट्वा प्रमेहं मधुरं सपिच्छं मधूपमं स्यादिद्ववधो विचारः ।
क्षीणेषु दोषेष्वनिलात्मकः स्यात् संतर्पणाद्वा कफसंभवः स्यात् ॥
C.S.Ci. 6/55

If *Prameha* patient passes urine which is sweet, slimy, and honey like, then there may be two possibilities i:e diminution of *Doṣās* leads to *Vātaja Prameha* and over nourishment leads to *Kaphaja Prameha.*

Doṣa : *Kapha Pradhāna Tridoṣaja*
Kapha → Kledaka
Pitta → Pācaka
Vāta → Vyāna & Apāna Vāta

Dūṣya : *Dhātu → Meda, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa.*
Updhātu →
Śārīrika Mala → Mūtra
Dhātu mala → Sveda, Keśa, Nakha, Ojas

Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni.*

Agniduṣṭi : *Mandāgni, Viṣamāgni* and *Tikṣnāgni* depending on predominance of *Kapha, Vāta & Pitta* respectively.

Āma : *Āgnijanya*

Srotas : *Mūtravaha Srotas*
Medavaha Srotas
Raktavaha Srotas
Śukravaha Srotas
Udakavaha Srotas
Majjāvaha Srotas
Rasavaha Srotas
Ojovaha Srotas
Māmsavaha Srotas

Srotoduṣṭi : *Atipravṛtti lakṣaṇa*

Udbhavasthāna : *Āmāśayottha, Pakvāśayottha*
Medovaha Srotasmūla

Sancārasthāna : *Rasāyani, Mūtravāhini, Rasavaha, Dhamani, Pittavaha Dhamani, Kaphavaha Dhamani, Śoṇitavaha Dhamani*

Vyaktasthāna : *Mūtravaha Srotas*

Adhiṣṭhāna : *Mūtravaha Srotas*

Svabhāva : *Āśukāri & Cirakāri*

Prabhāva : *Kaphaja-Curable*
Pittaja-Manageable
Vātaja-Incurable

Roga Bheda : 20 types (*Kaphaja 10, Pittaja 6 & Vātaja 4)*

Roga Mārga : *Madhyama Roga Mārga* and *Bāhya oga Mārga*

पूर्वरूप (Pūrvarupa)

स्वेदोऽङ्गगन्धः शिथिलाङ्गता च शय्यासनस्वप्नसुखे रतिश्च ।
हृन्नेत्रजिह्वाश्रवणोपदेहो घनाङ्गता केशनखातिवृद्धिः ॥
शीतप्रियत्वं गलतालुशोषो माधुर्यमास्ये करपाददाहः ।
भविष्यतो मेहगदस्य रूपं मूत्रेऽभिधावन्ति पिपीलिकाश्च ॥
C.S.Ci. 6/13-14

त्रयस्तु खलु दोषाः प्रकुपिताः प्रमेहानभिनिर्वर्तयिष्यन्त इमानि पूर्वरूपाणि दर्शयन्ति; तद्यथा-जटिलीभावं केशेषु, माधुर्यमास्यस्य, करपादयोः सुप्ततादाहौ, मुखतालुकण्ठशोषं, पिपासाम्, आलस्यं, मलं काये, कायच्छिद्रेषूपदेहं, परिदाहं सुप्ततां चाङ्गेषु, षट्पदपिपीलिकाभिश्च शरीरमूत्राभिसरणं, मूत्रे च मूत्रदोषान्,

विस्त्रं शरीरगन्धं, निद्रां, तन्द्रां च सर्वकालमिति ॥

C.S.Ni. 4/47

तेषां तु पूर्वरूपाणि-हस्तपादतलदाहः स्निग्धपिच्छिलगुरुता गात्राणां मधुरशुक्लमूत्रता तन्द्रा सादः पिपासा दुर्गन्धश्च श्वासस्तालुगलजिह्वादन्तेषु मलोत्पत्तिर्जटिलीभावः केशानां वृद्धिश्च नखानाम् ॥ *S.S.Ni. 6/5*

Aggravated all the three *Doṣās* develops following characteristic features during premonitory stage of the diseases : Matting of hairs, sweetishness in oral cavity, numbness and burning sensation in hands and feet, dryness in mouth, palate and throat, thirst, lassitude, excess accumulation of waste over the body especially in palate, throat, tongue & teeth, adherence of excreta in body orifices, burning sensation and numbness in body, accumulation of bees and ants over the body and urine, abnormality in the urine, fleshy smell from body, excess sleep and drowsiness. Excessive sweating, flaccidity of body, enjoys comfort in lying, sitting and sleeping, smearing in heart, eyes, tongue and ears, excessive growth of hair and nails, liking for cold substances, urine becomes sweet and whitish, breathlessness, heaviness, unctuousness and slimyness in the body.

रूप (Rupa)

प्रमेह सामान्य लक्षण (Prameha Sāmānya Lakṣaṇa)

प्रमेहपूर्वरूपाणामाकृतिर्यत्र दृश्यते ।
किञ्चिच्चाप्यधिकं मूत्रं तं प्रमेहिणमादिशेत् ॥
कृत्स्नान्यर्द्धानि वा यस्मिन् पूर्वरूपाणि मानवे ।
प्रवृद्धमूत्रमत्यर्थं तं प्रमेहिणमादिशेत् ॥

S.S.Ni. 6/22-23

Slight increase in quantity of urine associated with premonitory symptoms is known as patient of *Prameha.*

or

Complete appearance of premonitory symptoms or half of the premonitory symptoms associated with excess urination is known as patient of *Prameha.*

एवमेव दोषधातुमलाहारविशेषेणोत्कर्षापकर्षकृतेन संयोग-विशेषेण प्रमेहाणां नानाकरणं भवति ॥ *S.S.Ni. 6/26*

Increase or decrease of proportion in the combination of *Doṣās, Dhātus, Malās* and *Āhāra* are the main factors responsible for the development of various kinds of *Prameha.*

सामान्य लक्षण (Sāmānya Lakṣaṇa)

सामान्यं लक्षणं तेषां प्रभूताविलमूत्रता ।
दोषदूष्याविशेषेऽपि तत्संयोग विशेषतः ।
मूत्रवर्णादिभेदेन भेदो मेहेषु कल्प्यते ।

A.H.Ni. 10/7-8

Cardinal features of *Prameha* are turbidity and increased frequency. Though the *Doṣās* and *Dūṣyās* are similar, their combination being different, there occurs differences in colour etc. of urine based on these, different varieties of *Prameha* described.

Classification of Prameha

I.Two types of Prameha patient

द्दृष्ट्वा प्रमेहं मधुरं सपिच्छं मधूपमं स्याद्द्विविधो विचारः ।
क्षीणेषु दोषेष्वनिलात्मकः स्यात् संतर्पणाद्वा कफसंभवः स्यात् ।

C.S.Ci. 6/55

If the patient suffering from *Prameha* passes urine which is *Madhura* (sweet), *Picchila* (slimy) and *Madhupaṁa* (honey like), then there are two possibilities.

1. If it is caused by *Kṣiṇa Doṣa* (diminution of *Doṣās*) then it is considered as *Vāta* predominant. (*Vāta Pradhana Prameha*)
2. If it is caused by *Santarpaṇa* (over nourishment) then it is considered as *Kapha* predominant. (*Kapha Pradhana Prameha*)

II. Two types of Prameha patient

द्वौ प्रमेहौ भवतः-सहजोऽपथ्यनिमित्तश्च । तत्र सहजो मातृपितृबीजदोषकृतः, अहिताहारजोऽपथ्यनिमित्तः ।

S.S.Ci. 11/13

Prameha manifests due to two main etiological factors :

1. *Sahaja* (Hereditary/Congenital) - It manifests due to defective sperm and ovum of the father and mother respectively.
2. *Apathya nimittaja* (due to incompatible dietetics and activities)-It manifests due to

consumption of erratic dietetic pattern and activities.

III. Two types of Prameha patient

स्थूलः प्रमेही बलवानिहैकः कृशस्तथैकः परिदुर्बलश्च ।
संबृंहणं तत्रं कृशस्य कार्यं संशोधनं दोषबलाधिकस्य ॥

C.S.Ci. 6/15

Prameha is classified into two categories based on body constitution:

1. *Sthūla Prameh*ī - Those who are Sthula (obese) and *Balavāna* (strong).
2. *Kṛśa Prameh*ī- Those who are *Kṛṣa* (emaciated) and *Durbala* (weak).

IV. 20 Types of Prameha

त्रिदोषकोपनिमित्ता विंशतिः प्रमेहा भवन्ति ।

C.S.Ni. 4/3

साध्याः कफोत्था दश, पित्तजाः षट् याप्या, न साध्यः पवनाच्चतुष्कः । समक्रियत्वाद्विषमक्रियत्वान्महात्ययत्वाच्च यथाक्रमं ते ॥ *C.S.Ci. 6/7*

Simultaneous vitiation of all the three *Doṣās* manifest 20 types of *Prameha*. *Kaphaja Prameha* are ten in number and are curable because of the compatibility in the principles of treatment. *Pittaja Prameha* are six in number and are manageable because of the incompatibilities in the lines of treatment.

Vātaja Prameha are four in number and are incurable because of their severity of complications.

कफज प्रमेह हेतु (Kaphaja Prameha Hetu)

तत्रेमे त्रयो निदानादिविशेषाः श्लेष्मनिमित्तानां प्रमेहाणामाश्वभिनिर्वृत्तिकरा भवन्ति; तद्यथा–हायनकयवकचीनकोद्दालकनैषधेत्कटमुकुन्दकमहाव्रीहिप्रमोदकसुगन्धकानां नवानामतिवेलमतिप्रमाणेन चोपयोगः, तथा सर्पिष्मतां नवहरेणुमाषसूप्यानां, ग्राम्यानूपौदकानां च मांसानां, शाकतिलपललपिष्टान्नपायसकृशराविलेपीक्षुविकाराणां,क्षीरनवमद्यमन्दकदधिद्रवमधुरतरुणप्रायाणां चोपयोगः, मृजाव्यायामवर्जनं, स्वप्नशयनासनप्रसङ्गः, यश्च कश्चिद्विधिरन्योऽपि श्लेष्ममेदोमूत्रसंजननः स सर्वो निदानविशेषः ॥

C.S.Ni. 4/5

Special etiological factors for the manifestation of *Kapha prameha* are described are as follows:-

- Prolonged and excessive use of new grains like *Hāyanaka, Yavaka, Cīnaka, Uddālaka, Naiṣadha, Itkaṭa, Mukundaka, Mahāvrīhi, Pramodaka and Sugandhaka.*
- Consumption of new pulses like *hareṇu* and *Māṣa* with *Ghṛta.*
- Intake of meat of domestic, marshy and aquatic animals.
- Excess use of vegetables, sasamum paste, & flour preparations, *pāyasa, kṛśarā* (rice preparation with pulses and oil), *Vilepi* (gruel prepared with four times of water) and sugarcane products.
- Intake of milk, freshwine, immature curd and liquid substances, sweet and fresh substances, avoidance from cleanliness and physical exercises, indulging in excess sleep, bed rest and sedentary lifestyles.
- Indulgence of such factors which aggravate and vitiate *Kapha, Meda* and *Mūtra.*

कफज प्रमेह संप्राप्ति (Kaphaja Prameha Samprāpti)

बहुद्रवः श्लेष्मा दोषविशेषः ॥ बह्वबद्धं मेदो मांसं शरीरजक्लेदः शुक्रं शोणितं वसा मज्जा लसीका रसश्चौजःसंख्यात इति दूष्यविशेषाः ॥

त्रयाणामेषां निदानादिविशेषाणां सन्निपाते क्षिप्रं श्लेष्मा प्रकोपमापद्यते, प्रागतिभूयस्त्वात्; स प्रकुपितः क्षिप्रमेव शरीरे विसृप्तिं लभते, शरीर शैथिल्यात्; स विसर्पञ् शरीरे मेदसैवादितो मिश्रीभावं गच्छति, मेदसश्चैव बह्वबद्धत्वान्मेदसश्च गुणैः समानगुणभूयिष्ठत्वात्; स मेदसा मिश्रीभवन् दूषयत्येनत्, विकृतत्वात्; स विकृतो दुष्टेन मेदसोपहितः शरीरक्लेदमांसाभ्यां संसर्गं गच्छति, क्लेदमांसयोरतिप्रमाणाभिवृद्धत्वात्; स मांसे मांसप्रदोषात् पूतिमांसपिडकाः शराविकाकच्छपिकाद्याः संजनयति, अप्रकृतिभूतत्वात्; शरीरक्लेदं पुनर्दूषयन् मूत्रत्वेन परिणमयति, मूत्रवहानां च स्रोतसां वङ्क्षणबस्तिप्रभवाणां मेदःक्लेदोपहितानि गुरूणि मुखान्यासाद्य प्रतिरुध्यते; ततः प्रमेहांस्तेषां स्थैर्यमसाध्यतां वा जनयति, प्रकृतिविकृतिभूतत्वात् ॥ *C.S.Ni. 4/6-8*

Kapha is the main *Doṣa* consisting of abundant liquid associated with ten *Dūṣyās* namely *Medas* (which is abundant and non compact), *Māmsa, Śarīraja Kleda* (body fluids), *Śukra, Śoṇita, Vasā, Majjā, Laṣikā, Rasa*, and *Oja*. Aggravated *Kapha* due to etiological factors mixes with *Dūṣyās* and manifest *Kaphaja prameha*. Excacerbated *Kapha* spreads all over the body because of laxity of the body.

Kapha initially targets the *Medas* because it is abundantly increased, non-compact and similar in properties. As a result *Meda* gets vitiated. Both *Kapha* and *Meda* gets mixed with the body liquids and muscle tissue because latter two are increased excessively. This favour's the congenial atmosphere for the manifestation of putrefied carbuncles like *Śarāvika* and *Kacchapikā* etc. Liquid *Dhātus* further vitiated and transformed into urine. *Mūtravaha Srotas* gets obstructed due to application by *Meda* and liquid *Dhātus* of the body. All above consequences results in the manifestation of *Prameha,* which becomes either chronic or incurable. Stability or incurabilty is due to application of all the qualities of *Kapha* along with simultaneous vitiation of homogenous and heterogenous *dhātus.*

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kapha* consisting of abundant liquid

↓

vitiate *Dūṣyās i:e Medas, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa*

↓

Excacerbated *Kapha* spreads all over the body because of laxity of the body

↓

Both *doṣās and dūṣyās* localised in the *Mūtravaha Srotas*

↓

Kaphaja Prameha.

↓

Kapha initially targets the *Medas* because it is abundantly increased, non-compact and similar in properties

↓

As a result *Meda* gets vitiated.

↓

Both *Kapha* and *Meda* gets mixed with the body liquids and muscle tissue because latter two are increased excessively

↓

This favour's the congenial atmosphere for the manifestation of putrefied carbuncles like *Śarāvika* and *Kacchapikā* etc.

↓

Liquid *Dhātus* further vitiated and transformed into urine.

↓

Mūtravaha Srotas gets obstructed due to application by *Meda* and liquid *Dhātus* of the body.

↓

All above consequences results in the manifestation of *Kaphaja Prameha,* which becomes either chronic or incurable.

↓

Stability or incurabilty is due to application of all the qualities of *Kapha* along with simultaneous vitiation of homogenous and heterogenous *dhātus.*

कफज प्रमेह लक्षण (Kaphaja Prameha Lakṣaṇa)

शरीरक्लेदस्तु श्लेष्ममेदोमिश्रः प्रविशन् मूत्राशयं मूत्रत्वमापद्यमानः श्लैष्मिकैरेभिर्दशभिर्गुणैरुपसुज्यते वैषम्ययुक्तैः; तद्यथा– श्वेतशीतमूर्तपिच्छिलाच्छस्निग्धगुरुमधुरसान्द्रप्रसादमन्दैः, तत्र येन गुणेनैकेनानेकेन वा भूयस्तरमुपसृज्यते तत्समाख्यं गौणं नामविशेषं प्राप्नोति ॥ *C.S.Ni. 4/9*

All the liquid *dhātus* mixed with *Kapha* and *Meda* enter into the kidneys and bladder and get transformed into urine. Urine which is coming out is associated with ten morbid qualities of *Kapha* like whitishness, coldishness, viscosity, sliminess, transparent, unctuousness, heaviness, sweatiness, viscous, clear and slowness. Nomenclature of *Kaphaja Prameha* is done according to its association with one or more of the above ten properties of urine.

कफज प्रमेह भेद (Kaphaja Prameha Bheda)

ते तु खल्विमे दश प्रमेहा नामविशेषेण भवन्ति; तद्यथा– उदकमेहश्च, इक्षुवालिकारसमेहश्च, सान्द्रमेहश्च, सान्द्रप्रसादमेहश्च, शुक्लमेहश्च, शुक्रमेहश्च, शीतमेहश्च, सिकतामेहश्च, शनैर्मेहश्च, आलालमेहश्चेति ॥

ते दश प्रमेहाः साध्याः; समानगुणमेदःस्थानकत्वात्, कफस्य प्राधान्यात्, समक्रियत्वाच्च ॥ *C.S.Ni. 4/10-11*

Ten types of *Kaphaja Prameha* are described are as follows:-

1. *Udaka meha*
2. *Ikṣuvālikā rasa meha*
3. *Sāndra meha*
4. *Sāndra prasāda meha*
5. *Śukla meha*
6. *Śukra meha*
7. *Śīta meha*
8. *Sikatā meha*
9. *Śanair meha*
10. *Ālālameha*

All the above ten *Prameha* are curable because they are located in *Meda* having identical properties, the *Kapha* is predominant, and similarity in principles of treatment.

कफज प्रमेह विशेष लक्षण (Kaphaja Prameha Viśeṣa Lakṣaṇa)

1. उदकमेह (Udaka Meha)

अच्छं बहु सितं शीतं निर्गन्धमुदकोपमम्।
श्लेष्मकोपान्नरो मूत्रमुदमेही प्रमेहति ॥
C.S.Ni. 4/13

श्वेतमवेदनमुदकसदृशमुदकमेही मेहति । *S.S.Ni. 6/10*

Patients of *Udaka meha* passes urine, which is transparent, abundant, whitish, coldish, odourless and water like and patient passes urine without any discomfort.

2. इक्षुवालिका रस मेह (Ikṣuvālikā Rasa Meha)

अत्यर्थमधुरं शीतमीषत्पिच्छिलमाविलम्।
काण्डेक्षुरससङ्काशं श्लेष्मकोपात् प्रमेहति ॥
C.S.Ni. 4/14

Patients of *Ikṣuvālikā Rasa Meha* passes urine like sugarcane juice which is exceedingly sweet, cold, slightly saline and turbid.

3. सान्द्र मेह (Sāndra Meha)

यस्य पर्युषितं मूत्रं सान्द्रीभवति भाजने।
पुरुषं कफकोपेन तमाहुः सान्द्रमेहिनम् ॥
C.S.Ni. 4/15

आविलं सान्द्रं सान्द्रमेही । *S.S.Ni. 6/10*

Patients of *Sāndrameha* is diagnosed based on precipitation in the urine sample when kept over night, urine is turbid & thick.

4. सान्द्र प्रसाद मेह (Sāndra Prasāda Meha)

यस्य संहन्यते मूत्रं किंचित् किंचित् प्रसीदति।
सान्द्रप्रसादमेहीति तमाहुः श्लेष्मकोपतः ॥
C.S.Ni. 4/16

Qualities of the urine becomes partially viscous and partially clear when kept over night.

5. शुक्ल मेह (Śukla Meha)

शुक्लं पिष्टनिभं मूत्रमभीक्ष्णं यः प्रमेहति।
पुरुषं कफकोपेन तमाहुः शुक्लमेहिनम् ॥
C.S.Ni. 4/17

Patient of *Śuklameha* passes urine frequently which is white and flour like.

6. शुक्रमेह (Śukra Meha)

शुक्राभं शुक्रमिश्रं वा मुहुर्मेहति यो नरः।
शुक्रमेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः ॥
C.S.Ni. 4/18

Patients of *Śukrameha* passes urine frequently, which is like semen or mixed with semen.

7. शीतमेह (Śīta Meha)

अत्यर्थमधुरं शीतं मूत्रं मेहति यो भृशम्।
शीतमेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः ॥
C.S.Ni. 4/19

Patients of *Śītameha* passes urine oftenly which is sweet & cold.

8. सिकता मेह (Sikatā Meha)

मूर्तान्मूत्रगतान् दोषानणून्मेहति यो नरः।
सिकतामेहिनं विद्यात्तं नरं श्लेष्मकोपतः ॥
C.S.Ni. 4/20

सरुजं सिकताऽनुविद्धं सिकतामेही । *S.S.Ni. 6/10*

Patients of *Sikatāmeha* passes urine associated with small particles (gravel) and pain.

9. शनैर्मेह (Śanair Meha)

मन्दं मन्दमवेगं तु कृच्छ्रं यो मूत्रयेच्छनैः।
शनैर्मेहिनमाहुस्तं पुरुषं श्लेष्मकोपतः ॥
C.S.Ni. 4/21

सकफं मृत्स्नं शनैर्मेही । *S.S.Ni. 6/10*

Patient of *Śanairmeha* passes urine with difficulty and slowly without any urge. Urine appears like mixed with *Kapha* and slimy.

10. आलाल मेह (Ālāla Meha)

तन्तुबद्धमिवालालं पिच्छिलं यः प्रमेहति।
आलालमेहिनं विद्यात्तं नरं श्लेष्मकोपतः॥
C.S.Ni. 4/22

Patient of *Ālālameha* passes urine which appears like full of threads, saliva like and slimy.

Śusruta described following ten varieties of *Kaphaja Prameha* are as follows:-

कफादुदकेक्षुवालिकासुरासिकताशनैर्लवणपिष्टसान्द्रशुक्रफेनमेहा दश साध्याः । *S.S.Ni. 6/8*

1. *Udaka meha*
2. *Ikṣuvālikā meha*
3. *Surā meha*
4. *Sikatā meha*
5. *Śanair meha*
6. *Lavaṇa meha*
7. *Piṣṭa meha*
8. *Sāndra meha*
9. *Śukra meha*
10. *Phena meha*

सुरा मेह (Surā Meha)

सुरामेही सुरातुल्यम् । *S.S.Ni. 6/10*

Patients of *Surā meha* passes urine resembling *Surā* (beer).

लवण मेह (Lavaṇa Meha)

विशदं लवणतुल्यं लवणमेही । *S.S.Ni. 6/10*

Patient of *Lavaṇa meha* passes urine which is non slimy and resembling saltish taste.

पिष्ट मेह (Piṣṭa Meha)

दृष्टरोगा पिष्टरसतुल्यं पिष्टमेहा । *S.S.Ni. 6/10*

Patients of *Piṣṭa meha* passes urine which is flour like associated with horripilations.

फेन मेह (Phena Meha)

स्तोकं स्तोकं सफेनं फेनमेही मेहति ॥ *S.S.Ni. 6/10*

Patients of *Phena meha* passes little urine often which is frothy and clear.

पित्तज प्रमेह हेतु (Pittaja Prameha Hetu)

उष्णाम्ललवणक्षारकटुकाजीर्णभोजनोपसेविनस्तथाऽतितीक्ष्णातपाग्निसंतापश्रमक्रोधविषमाहारोपसेविनश्च तथाविधशरीरस्यैव क्षिप्रं पित्तं प्रकोपमापद्यते, तत्तु प्रकुपितं तयैवानुपूर्व्या प्रमेहानिमान् षट् क्षिप्रतरमभिनिर्वर्तयति ॥ *C.S.Ni. 4/24*

The following etiological factors are responsible for the manifestation of *Pittaja Prameha* : One who consumes substances which are very hot, sour, saline, alkaline and pungent; intake of food before the digestion of previous meal; excessive exposure to intense heat of the sun, fire, physical exertion and anger; intake of incompatible dietetics.

Pittaja Prameha Samprāpti

The aggravated *Pitta* due to above causes enters *Medas, Māmsa, Śarīraja Kleda, Śukra, Śoṇita, Vasā, Majjā, Lasikā, Rasa* and *Oja* leading to development of *Pittaja Prameha.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Pitta*
↓
vitiate *Dūṣyās i:e Medas, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa*
↓
Excacerbated *Pitta and Kapha* spreads all over the body because of laxity of the body.
↓
Both *doṣās and dūṣyās* localised in the *Mūtravaha Srotas*
↓
Pittaja Prameha.

Pittaja Prameha is classified into six varieties-

1. *Kṣāra meha*
2. *Nīla meha*
3. *Mañjiṣṭā meha*
4. *Kāla meha*
5. *Rakta meha*
6. *Haridā meha*

साध्यासाध्यता एवं पित्तज प्रमेह लक्षण (Sādhyāsādhyatā evam Pittaja Prameha Lakṣaṇa)

सर्व एव ते याप्याः, संसृष्टदोषमेदःस्थानत्वाद्विरुद्धोपक्रमत्वाच्चेति । तत्र श्लोकाः पित्तप्रमेहविशेषविज्ञानार्था भवन्ति । इत्येते षट् प्रमेहाः पित्तप्रकोपनिमित्ता व्याख्याता भवन्ति ॥
C.S.Ni. 4/27,28,35

Pittaja Pramehās are manageable due to antagonicity in the lines of treatment and involvement of multiple *Doṣās*. The treatment for vitiated *Pitta* and *Medas* involves mutual contradiction.

1. क्षार मेह (Kṣāra Meha)

गन्धवर्णरसस्पर्शैर्यथा क्षारस्तथाविधम् ।
पित्तकोपान्नरो मूत्रं क्षारमेही प्रमेहति ॥
C.S.Ni. 4/29

Patient of *Kṣāra meha* passes urine having the smell, colour, taste and touch like that of alkalies.

2. काल मेह (Kāla Meha)

मसीवर्णमजस्त्रं यो मूत्रमुष्णं प्रमेहति ।
पित्तस्य परिकोपेण तं विद्यात् कालमेहिनम् ॥
C.S.Ni. 4/30

Patient of *Kāla meha* passes large amount of urine, which is blackish and hot.

3. नील मेह (Nīla Meha)

चाषपक्षनिभं मूत्रमम्लं मेहति यो नरः ।
पित्तस्य परिकोपेण तं विद्यान्नीलमेहिनम् ॥
C.S.Ni. 4/31

Patient of *Nīla meha* passes urine as sour and having colour like that of the feather of *Cāsa* bird (blue jay).

4. रक्त मेह (Rakta Meha)

विस्त्रं लवणमुष्णं च रक्तं मेहति यो नरः ।
पित्तस्य परिकोपेण तं विद्याद्रक्तमेहिनम् ॥
C.S.Ni. 4/32

Patient of *Rakta meha* passes urine as having fleshy smell, saline, hot and red.

5. मञ्जिष्ठ मेह (Mañjiṣṭha Meha)

मञ्जिष्ठोदकसंकाशं भृशं विस्त्रं प्रमेहति ।
पित्तस्य परिकोपात्तं विद्यान्माञ्जिष्ठमेहिनम् ॥
C.S.Ni. 4/33

Patient of *Mañjiṣṭha meha* passes urine frequently resemble like decoction of *Mañjiṣṭa* having the smell like that of raw flesh.

6. हारिद्र मेह (Hāridra Meha)

हरिद्रोदकसङ्काशं कटुकं यः प्रमेहति ।
पित्तस्य परिकोपात्तं विद्याद्धारिद्रमेहिनम् ॥
C.S.Ni. 4/34

सदाहं हरिद्राऽऽभं हरिद्रामेही । *S.S.Ni. 6/11*

Patient of *Hāridrameha* passes urine resemble like decoction of *haridra* having pungent taste accompanied with burning sensation.

वातज प्रमेह हेतू (Vātaja Prameha Hetu)

कषायकटुतिक्तरूक्षलघुशीतव्यवायव्यायामवमनविरेचनास्थापनशिरोविरेचनातियोगसंधारणानशनाभिघातातपोद्वेगशोकशोणितातिषेकजागरणविषमशरीरन्यासानुपसेवमानस्य तथाविधशरीरस्यैव क्षिप्रं वातः प्रकोपमापद्यते ॥ *C.S.Ni. 4/36*

The following etiological factors are responsible for the manifestation of *Vātaja prameha.*

Excessive consumption of astringent, pungent, bitter, rough, light and cold things; excessive sexual intercourse and physical exercises; excess administration of enema, purgation, non-unctuous enema, and head evacuation; suppression of natural urges, fasting, trauma, exposure to sun, anxiety, grief, excessive blood letting, keeping awake at night, and uneven body postures.

वातज प्रमेह संप्राप्ति (Vātaja Prameha Samprāpti)

स प्रकुपितस्तथाविधे शरीरे विसर्पन् यदा वसामादाय मूत्रवहानि स्त्रोतांसि प्रतिपद्यते तदा वसामेहमभिनिर्वर्तयति; यदा पुनर्मज्जानं मूत्रवस्ता-वाकर्षति तदा मज्जमेहमभिनिर्वर्तयति; यदा तु लसीकां मूत्राशयेऽभिवहन्मूत्रमनुबन्धं च्योतयति लसीकातिबहुत्वाद्विक्षेपणाच्च वायोः खल्वस्यातिमूत्रप्रवृत्तिसङ्गं करोति, तदा स मत्त इव गजः क्षरत्यजस्त्रं मूत्रमवेगं, तं हस्तिमेहिनमाचक्षते; ओजः पुनर्मधुरस्वभावं, तद् यदा रौक्ष्याद्वायुः कषायत्वेनाभिसंसृज्य मूत्राशयेऽभिवहति तदा मधुमेहं करोति ॥ *C.S.Ni. 4/37*

1. वसा मेह (Vasā Meha)

When aggravated *Vāta* spreading all over the body mixes with *Vasā* and enters into the *Mūtravaha Srotas* and manifest *Vasāmeha.*

2. मज्जा मेह (Majjā Meha)

If aggravated *Vāta* carries *Majjā* to the *Mutravaha Srotas* leading to manifestation of *Majjā meha.*

3. हस्ति मेह (Hasti Meha)

When aggravated *Vāta* mixes with *Lasikā* enters into *Mūtravaha Srotas* leading to manifestation of *Lasikāmeha.* It is characterised by continuous excretion of urine due to abundance of *Lasikā* and frequent micturition with obstruction due to dispersing nature and patient passes large amount of urine continuously without any urge like an excited elephant, that's why, it is called *Hastimeha*.

4. मधुमेह (Madhumeha)

Sweetish nature of *Ojas* gets converted into *Astringent* taste by aggravated *Vāta* and carries into the *Mūtravaha Srotas* and manifest ***Madhumeha.***

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vāta*
↓
vitiate *Dūṣyās i:e Medas, Rakta, Śukra, Ambu, Vasā, Lasikā, Majjā, Rasa, Ojas* and *Māmsa*
↓
Excacerbated *Vāta and Kapha* spreads all over the body because of laxity of the body
↓
Both *doṣās and dūṣyās* localised in the *Mūtravaha Srotas*
↓
Vātaja Prameha.

साध्यासाध्यता एवं वातज प्रमेह लक्षण (Sādhyāsādhyatā evam Vātaja Prameha Lakṣaṇa)

इमांश्चतुरः प्रमेहान् वातजानसाध्यानाचक्षते भिषजः, महात्ययिकत्वाद्विरुद्धोपक्रमत्वाच्चेति ॥

तेषामपि पूर्ववद्गुणविशेषेण नामविशेषा भवन्ति; तद्यथावसामेहश्च, मज्जमेहश्च, हस्तिमेहश्च, मधुमेहश्चेति ॥

तत्र श्लोका वातप्रमेहविशेषविज्ञानार्था भवन्ति॥

C.S.Ni. 4/38-40

Vātaja Pramehās are said to be incurable due to their severity and *antagonisity* in the lines of treatment.

It is subclassified into 4 varieites are as follows:-

1. *Vasā meha*
2. *Majjā meha*
3. *Hasti meha*
4. *Madhu meha*

Signs and symptoms indicating poor prognosis in *Vātaja Prameha*

श्यावारुणो वातकृतः सशूलो मज्जादिसाद्गुण्यमुपैत्यसाध्यः ॥

C.S.Ci. 6/12

Vātaja Pramehās are characterised by greyish or reddish discoloration of urine associated with pain and similarity with *Majjā* etc are incurable.

वातज प्रमेह विशेष लक्षण (Vātaja Prameha Viśeṣa Lakṣaṇa)

वसा मेह (*Vasā Meha*)

वसामिश्रं वसाभं वा मुहुर्मेहति यो नरः ।
वसामेहिनमाहुस्तमसाध्यं वातकोपतः ॥

C.S.Ni. 4/41

It is said to be incurable and it manifest due to predominance of *Vāta* and patient passes urine frequently mixed with *Vasā* or having the resemblance like that of *Vasā.*

मज्जा मेह (Majjā Meha)

मज्जानं सह मूत्रेण मुहुर्मेहति यो नरः ।
मज्जमेहिनमाहुस्तमसाध्यं वातकोपतः ॥

C.S.Ni. 4/42

Patient of *Majjā meha* passes urine frequently mixed with *Majjā*. It is incurable.

हस्ति मेह (Hasti Meha)

हस्ती मत्त इवाजस्त्रं मूत्रं क्षरति यो भृशम् ।
हस्तिमेहिनमाहुस्तमसाध्यं वातकोपतः ॥

C.S.Ni. 4/43

Patient of *Hastimeha* passes urine excessively and constantly like an excited elephant, and it is incurable.

साध्यासाध्याता (Sādhyā Sādhyatā)

सपूर्वरूपाः कफपित्तमेहाः क्रमेण ये वातकृताश्च मेहाः । साध्या न ते, पित्तकृतास्तु याप्याः, साध्यास्तु मेदो यदि न प्रदुष्टम् ॥

C.S.Ci. 6/56

Kaphaja and *Paittika* types of *Prameha* are incurable if premonitory symptoms persist. *Vātaja* types of Prameha are incurable. *Pittaja* types of *Prameha* are manageable. All kinds of *Pramehās* are curable if *Meda dhātu* is not vitiated.

मधुमेह (Madhumeha)

मधुमेह हेतु (Madhumeha Hetu)

गुरुस्निग्धाम्ललवणान्यतिमात्रं समश्नताम् ।
नवमन्नं च पानं च निद्रामास्यासुखानि च ॥
त्यक्तव्यायामचिन्तानां संशोधनमकुर्वताम् ।
C.S.Su.17/78-79

The following etiological factors are responsible for the development of *Madhumeha*. Excessive intake of heavy, unctuous, sour and saline taste foods.

Newly harvested crops like cereals, rice etc. and fresh wine consumption in large quantity. Sedentary life-style, not indulging in any sort of physical and mental exercise. One who does not undergo bio-purification of the body.

मधुमेह संप्राप्ति (Madhumeha Samprāpti)

श्लेष्मा पित्तं च मेदश्च मांसं चातिप्रवर्धते ॥
तैरावृतगतिर्वायुरोज आदाय गच्छति ।
यदा बस्तिं तदा कृच्छ्रो मधुमेहः प्रवर्तते ॥
स मारुतस्य पित्तस्य कफस्य च मुहुर्मुहुः ।
दर्शयत्याकृतिं गत्वा क्षयमाप्यायते पुनः ॥
उपेक्षयाऽस्य जायन्ते पिडकाः सप्त दारुणाः ।
मांसलेष्ववकाशेषु मर्मस्वपि च संधिषु ॥
C.S.Su.17/79-82

All above etiological factors aggravates *Kapha, Pitta, Meda* and *Māmsa* and obstruct the normal pathway of *Vāta*. Agitated *Vāta* carries the *Ojas* to the basti i:e *Mūtravaha Srotas* and manifest *Madhumeha,* which is difficult for management.

It manifest the signs and symptoms pertaining to *Vāta, Pitta* and *Kapha* quite oftenly and these symptomatology may disappear at time and reappear again at appropriate time. Negligence in the management of this disease leads to development of seven severe *Piḍakās* over muscular areas, vital parts and joints.

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kapha, Pitta, Meda* and *Māmsa*

↓

Obstruct the normal pathway of *Vāta*

↓

Agitated *Vāta* carries the *Ojas* to the basti i:e *Mūtravaha Srotas*

↓

Madhumeha (which is difficult for management)

↓

It manifest the signs and symptoms pertaining to *Vāta, Pitta* and *Kapha* quite oftenly

↓

and these symptomatology may disappear at time and reappear again at appropriate time.

↓

Negligence in the management of this disease

↓

leads to development of seven severe *Piḍakās* over muscular areas, vital parts and joints.

मधुमेह लक्षण (Madhumeha Lakṣaṇa)

कषायमधुरं पाण्डु रूक्षं मेहति यो नरः ।
वातकोपादसाध्यं तं प्रतीयान्मधुमेहिनम् ॥
C.S.Ni. 4/44

Patient of *Madhumeha* passes urine which is astringent, sweet, pale and ununctous and it is incurable.

सर्व एव प्रमेहा मूत्रादिमाधुर्ये मधुगन्धसामान्यात् पारिभाषिकीं मधुमेहाख्यां लभन्ते ॥ *S.S.Ci. 12/6*

Madhumeha patients passes urine having sweet in taste and smell of the body resembling like honey.

कुलज मधुमेह (Kulaja Madhumeha)

जातः प्रमेही मधुमेहिनो वा न साध्य उक्तः स हि बीजदोषात्।
ये चापि केचित् कुलजा विकारा भवन्ति तांश्च प्रवदन्त्यसाध्यान् ॥
C.S.Ci. 6/57

Patients of *Prameha* and *Madhumeha* if develops right from the time of birth or patients of *Prameha/Madhumeha*, who is born of parents who were also suffering from the same ailments are said to be incurable because of the morbidity in their Bija (genes). In the same way other inherited disorders are also considered as incurable.

सर्व एव प्रमेहास्तु कालेनाप्रतिकारिणः ।
मधुमेहत्वमायान्ति तदाऽसाध्या भवन्ति हि ॥
S.S.Ni. 6/27

If treatment is not employed to all varieties of

Prameha at the appropriate time converted to *Madhumeha* and it becomes incurable.

मधुमेहो मधुसमं, जायते स किल द्विधा ॥
क्रुद्धे धातुक्षयाद्वायौ दोषावृतपथेऽथवा।
आवृतो दोषलिङ्गानि सोऽनिमित्तं प्रदर्शयेत् ॥
क्षीणः क्षणात्क्षणात् पूर्णो भजते कृच्छ्रसाध्यताम्।
कालेनोपेक्षिताः सर्वे यद्यान्ति मधुमेहताम् ॥
मधुरं यच्च सर्वेषु प्रायो मध्विव मेहति।
सर्वेऽपि मधुमेहाख्या माधुर्याच्च तनोरतः ॥
A.H.Ni. 10/18-21

Disease *Madhumeha* is characterised by excretion of urine in excess which resembles like honey. It arises due to two main reasons i:e aggravation of *Vāta* by *Dhātukṣaya* and obstruction of its path by the *Doṣās* covering it.

Mārgāvṛtajanya Madhumeha

It manifest the symptoms of the *Doṣa* covering it.

Dhātukṣayajanya Madhumeha

In case if *Madhumeha* develops due to *Dhātukṣaya* it becomes difficult to cure without any apparent cause, bladder becomes empty sometimes and full some times.

All varieties of *Prameha,* if neglected, get converted to *Madhumeha* in due course of time. Characteristic features of *Madhumeha* is the execretion of the urine in excess, which resembles like honey and the whole body also becoming sweet.

उपद्रव (Upadrava)

उपद्रवास्तु खलु प्रमेहिणां तृष्णातीसारज्वरदाहदौर्बल्यारोचकाविपाकाः पूतिमांसपिडकालजी-विद्रध्यादयश्च तत्प्रसङ्गाद्भवन्ति ॥ *C.S.Ni. 4/48*

Complications of *Prameha* are thirst, diarrhoea, fever, burning sensation, weakness, anorexia & indigestion, development of various *Prameha Piḍakās,* and putrification of muscle tissue leads to *Alaji* and *Vidradhi* during the chronic stage of the disease.

विशेष उपद्रव (Viśeṣa Upadrava)

मक्षिकोपसर्पणमालस्यं मांसोपचयः प्रातिश्यायः शैथिल्यारोचकाविपाकाः कफप्रसेकच्छर्दिनिद्राकास-श्वासाश्चेति श्लेष्मजानामुपद्रवाः, वृषणयोरवदरणं बस्तिभेदो मेढ्रतोदो हृदि शूलमम्लिकाज्वरातीसारारोचका वमथुः परिधूमायनं दाहो मूर्च्छा पिपासा निद्रानाशः पाण्डुरोगः पीतविण्मूत्रनेत्रत्वं चेति पैत्तिकानाम्। हृद्ग्रहो लौल्यमनिद्रा स्तम्भः कम्पः शूलं बद्धपुरीषत्वं चेति वातजानाम्। एवमेते विंशतिः प्रमेहाः सोपद्रवाव्याख्याताः ॥

S.S.Ni. 6/15

Upadrava develops in all *Doṣic* types of *Prameha* are as follows:-

***Kaphaja Prameha Upadrava* :** Flies swarm towards the body, lassitude, slight development of muscle tissue, nasal catarrah, looseness of body parts, loss of taste, indigestion, excessive salivation, vomiting, excess sleep, cough and breathlessness.

Pittaja Prameha Upadrava : Appearance of cracks over scrotum, pain in the bladder, penis and heart, sour belching, fever, diarrhoea, loss of taste, vomiting, patient feels as if smoke is coming out from the mouth, burning sensation, fainting, thirst, loss of sleep, pallor, yellowish discolouration of faeces, urine and eyes.

Vātaja Prameha Upadrava : Feeling of stiffness in cardiac region, excess greed for all things, loss of sleep, rigidity, tremors, pain in abdomen and constipation.

प्रमेह पिडका (Prameha Piḍaka)

तत्र वसामेदोभ्यामभिपन्नशरीरस्य त्रिभिर्दोषैश्चानुगतधातोः प्रमेहिणो दश पिडका जायन्ते। तद्यथा-शराविका, सर्षपिका, कच्छपिका, जालिनी, विनता, पुत्रिणी, मसूरिका, अलजी, विदारिका, विद्रधिका चेति ॥ *S.S.Ni. 6/16*

Prameha Piḍakās develop in such patients in whom *Vasā* and *Meda* are excessively accumulated in the body and these are invaded by abnormal three *Doṣās* leading to development of ten kinds of *Prameha Piḍakās* namely *Śarāvika, Sarṣapikā, Kacchapikā, Jālinī, Vinatā, Putriṇī, Masūrikā, Alajī, Vidārikā* and *Vidradikā. Caraka Samhitā* described seven varieties of *Piḍakās* are as follows.

शराविका कच्छपिका जालिनी सर्षपी तथा।
अलजी विनताख्या च विद्रधी चेति सप्तमी ॥
C.S.Su. 17/83

Śarāvikā, *Kacchapikā*, *Jālini*, *Sarṣapī*, *Alajī*, *Vinatā* and *Vidradhī*.

शराविका (Śarāvikā)

अन्तोन्नता मध्यनिम्ना श्यावा क्लेदरुगन्विता।
शराविका स्यात् पिडका शरावाकृतिसंस्थिता॥
C.S.Su. 17/84

शरावमात्रा तद्रूपा निम्नमध्या शराविका।
S.S.Ni. 6/17

Śarāvikā type of *Prameha Piḍaka* characterised by development of carbuncles which are raised at margins and depressed at the centre; blackish discolouration, associated with moisture and pain and it resemble like that of earthen saucer.

कच्छपिका (Kacchapikā)

अवगाढार्तिनिस्तोदा महावास्तुपरिग्रहा।
श्लक्ष्णा कच्छपपृष्ठाभा पिडका कच्छपी मता॥
C.S.Su. 17/85

सदाहा कूर्मसंस्थाना ज्ञेया कच्छपिका बुधैः।
S.S.Ni. 6/16

Kacchapikā type of carbuncle is deep seated associated with severe pain, burning sensation extends to broad area, smooth and resembles like the back of tortoise.

जालिनी (Jālinī)

स्तब्धा सिराजालवती स्निग्धास्रावा महाशया।
रुजानिस्तोदबहुला सूक्ष्मच्छिद्रा च जालिनी॥
C.S.Su. 17/86

जालिनी तीव्रदाहा तु मांसजालसमावृता॥
S.S.Ni. 6/18

Jālinī variety of *Prameha Piḍaka* is very stiff, surrounded by network of blood vessels, unctuous discharge having deep base associated with various kinds of pain and burning sensations along with subtle opening.

सर्षपी (Sarṣapī)

पिडका नातिमहती क्षिप्रपाका महारुजा।
सर्षपी सर्षपाभाभिः पिडकाभिश्चिता भवेत्॥
C.S.Su. 17/87

गौरसर्षपसंस्थाना तत्प्रमाणा च सर्षपी॥
S.S.Ni. 6/15

Sarṣapī type of carbuncle is not very big, suppurates quickly associated with severe pain and its size is similar to white mustard seeds.

अलजी (Alajī)

दहति त्वचमुत्थाने तृष्णामोहज्वरप्रदा।
विसर्पत्यनिशं दुःखाद्दहत्यग्निरिवालजी॥
C.S.Su. 17/88

रक्ता सिता स्फोटवती दारुणा त्वलजी भवेत्॥
S.S.Ni. 6/18

It is characterised by burning sensation in skin during its appearance of red or white coloured vesicle associated with thirst, unconsciousness and fever. It spreads constantly and produces difficult situation by inducing terrible burning sensation like fire. Vesicles may burst leading to dreadful condition.

विनता (Vinatā)

अवगाढरुजाक्लेदा पृष्ठे वाऽप्युदरेऽपि वा।
महती विनता नीला पिडका विनता मता॥
C.S.Su. 17/89

महती पिडका नीला पिडका विनता स्मृता।
S.S.Vi. 6/19

Vinatā variety of *Prameha Piḍakā* is deep seated associated with pain and thick discharge. It manifests either in back or abdomen and it is bigger in size having bluish discoluration and depression at the centre.

पुत्रिणी (Putriṇī)

महत्यल्पाचिता ज्ञेया पिडका सा तु पुत्रिणी॥
S.S.Ni. 6/19

It is bigger in size sorrounded by many smaller eruptions.

मसूरिका (Masūrikā)

मसूरसमसंस्थाना ज्ञेया या तु मसूरिका।
S.S.Ni. 6/20

Appearance of skin eruptions resemble like lentil size and shape

विदारिका (Vidārikā)

विदारीकन्दवद् वृत्ता कठिना च विदारिका।
S.Su.Ni. 6/21

It resembles like *Vidārīkanda* and is hard.

विद्रधिका (Vidradhikā)

विद्रधेर्लक्षणैर्युक्ता ज्ञेया विद्रधिका बुधैः॥
S.S.Ni. 6/21

Features are similar like that of *Vidradhi.*

Note : If these *Piḍakās* develop in the anus/rectum, heart, head, shoulders, back and vital organs associated with complications and poor digestive capacity should be rejected for treatment. *(S.S.Ni. 6/20).*

उपशय/पथ्य (Upaśaya/Pathya)

C.Ci. 6th Chapter, S.S.Ci. 10, 11 & 12th Chapter

औषध (Auṣadha)

Kaphaja Prameha

Haritakyādi Kvātha
Haridrādi Kvātha
Cavyādi Kvātha etc.

Pittaja Prameha

Uśīrādi Kvātha
Nimbādi Kvātha
Mustādi Kvātha etc.

Vātaja Prameha

Tindukādi Kvātha
Triphalādi Kvātha
Agnimantha Kvātha etc

Common medicines-

Madhvāsava
Bhallātakāsava
Mākṣika Dhātu Yoga
Navāyasa Lauha
Dantyāsava
Śilājatu Yoga
Śālasārādi Avaleha
Lohāriṣṭa

अन्न (Anna)

सारोदकं वाऽथ कुशोदकं वा मधूदकं वा त्रिफलारसं वा।
सीधुं पिबेद्वा निगदं प्रमेही माध्वीकमग्र्यं चिरसंस्थितं वा॥
मांसानि शूल्यानि मृगद्विजानां खादेद्यवानां विविधांश्च भक्ष्यान्।
संशोधनारिष्टकषायलेहैः संतर्पणोत्थञ् शमयेत् प्रमेहान्॥
भृष्टान् यवान् भक्षयतः प्रयोगाच्छुष्कांश्च सक्तून्न भवन्ति मेहाः।
श्वित्रं च कृच्छ्रं कफजं च कुष्ठं तथैव मुद्गामलकप्रयोगात्॥
C.S.Ci. 6/46-48

Patients of *Prameha* should drink *Sārodaka, Kuśodaka, Madhūdaka, Triphalārasa, Sīdhu, Mādvika.*

Patient should eat roasted meat of animals and birds and various preparations of barley. Patients of *Prameha* caused by over saturation should be alleviated by the administration of elimination therapies, *Ariṣṭās*, decoctions and linctus.

Habitualy consumption of parched barley, dry corn flour, *Mudga* and *Āmalaka* do not suffer from *Prameha, Śvitra* and *Kṛcchra Kaphaja Kuṣṭha.*

ततः शालिषष्टिकयवगोधूमकोद्रवोद्दालकाननवान् भुञ्जीत चणकाढकीकुलत्थमुद्गविकल्पेन, तिक्तकषायाभ्यां च शाकगणाभ्यां निकुम्भेङ्गुदीसर्षपातसीतैलसिद्धाभ्यां, बद्धमूत्रैर्वा जाङ्गलैर्मांसैरपहृतमेदोभिरनम्लैरघृतैश्चेति॥ *S.S.Ci. 11/6*

One should consume food prepared from old *Śāli, Ṣaṣṭika, Yava, Godhūma, Kodrava* or *Uddālaka* along with soup prepared either by *Caṇaka, Āḍhaki, Kulattha & Mudga, Vegetables* of bitter and astringent taste, groups of vegetables and oils of *Nikumbha, Iṅgudi, Sarṣapa* and *Atasī.*

During obstruction to maturation patient should eat fat free meat of animals of desert animals without adding sour and ghee.

विहार (Vihāra)

प्रवृद्धमेहास्तु व्यायामनियुद्धक्रीडागजतुरगरथपदातिचर्यापरिक्रमणान्यस्त्रोपास्त्रे वा सेवेरन्॥ परिक्रमणं चङ्क्रमणं, गतागतामेत्यर्थः, सर्वतो भ्रमणमित्यन्ये। डल्हणः।

अधनस्त्वबान्धवो वा पादत्राणातपत्रविरहितो भैक्ष्याशी ग्रामैकरात्रवासी मुनिरिव संयतात्मा योजन शतमधिकं वा गच्छेत्, महाधनो वा श्यामाकनीवारवृत्तिरामलककपित्थतिन्दुकाश्मन्तकफलाहारो मृगैः सह वसेत्, तन्मूत्रशकृद्भक्षः सततमनुव्रजेद्गाः, ब्राह्मणो वा शिलोञ्छवृत्तिर्भूत्वा ब्रह्मरथमुद्धरेत्, कृषेत् सततमितरः खनेद्वा कूपं, कृशं तु सततं रक्षेत्॥ *S.S.Ci. 11/11-12*

During advanced stage of the disease patient should indulge in physical exercises, wrestling, sports, riding on elephant, horses or chariot, foot exercises, briskwalk, archery etc. daily.

One who is poor should undertake 100 Yojana journey on foot, without taking money or relatives with him without using foot wear and umbrella. Eat food obtained through begging staying at one village for one day, leading a life of an ascetic and remaining self controlled.

One who is rich should consume food prepared from *Śyāmāka* or *Nīvāra* along with intake of fruits like *Āmalaka, Kapittha, Tinduka,* and *Aśmantaka* and stay in the company of animals, consume their urine and dung and move along with them always. Patients who are *Brāhmaṇa* should adopt the profession of sculptor and carve a *Brahmaratha.* Others should indulge constantly in ploughing fields or digging wells. Emaciated *Prameha* patient should protect his body constantly by adopting appropriate procedures.

अधनो वैद्यसन्देशादेवं कुर्वन्नतन्द्रितः ।
संवत्सरादन्तराद्वा प्रमेहात् प्रतिमुच्यते ॥
S.S.Ci. 11/13

Patient who is poor, adhering to the above regimen as per physician's advise without any lassitude gets cured of *Prameha* within one year or within that period.

In *Prameha,* the foodstuffs like *Mantha,* sour, juice, powder and ointments made with meat soups of *Viṣkira* (ones having habits of eating wasteful products) and *Pratuda* (ones having long beaks) birds as well as of the wild animals is considered to be a good diet. In addition, lentil, *Mudga* and similar light edibles are also recommended for a *Prameha* patient. (B.R. 37/3)

Diet and Regimens: For the patient suffering from *Prameha* food ingredients containing *Śyāmaka, Kodrava* and *Uddālaka* variety of rice, *Godhūma* (wheat), *Caṇaka, Ādhaki* and *Kulattha* pulse beans which are stored for a long time after harvesting are always wholesome. He should take meat of *Jāṅgala* animals (inhabiting arid land), vegetables having bitter taste, barley and honey. He should perform physical exercise. (B.R. 37/4)

Purification of the body through such bitter herbs like *Neem* and *Haridrā* should be applied to the patient. He should also take up exercises and should remain awake upto late night hours. The herbs which controls *Kapha* and *Pitta* should be applied in massaging his body. Such external as well as internal measures would provide relief to the patient. (B.R. 37/5)

All types of exercises cure *Prameha.* The patient should continuously walk for one hundred *Yojanās* without using shoes and umbrella, and by begging for his food like a saint. Optionally, he should reside in a forest by eating *Nīvāra* and *Āmalakī* which will make him free from the disease. (B.R. 37/15-16)

Once the symptoms of *Prameha* disease are clear, one should first have *Laṅgana* (fasting), vomiting, *Virecana* (purgation) therapies. Apart from this, one should also take appetizer meals and herbs. Massaging should also be applied to the patient. The following eatables are helpful in case of *Prameha*: *Nivāra*, *Kangu*, barley, soft bamboos, wild variety of *Kulattha*, *Mukundaka* variety of rice, old wheat grains, *Śāli* variety of rice, *Kalma* variety of rice, *Yūṣa* of pulses of the *Kulattha*, *Mudga* and pigeon pea, *Lājā* of paddy, old liquor, honey, *Vaṭyamaṇḍa* (*Maṇḍa* prepared by cooking barley in water measuring four times), buttermilk, urine of an ass, urine of a buffalo, pigeon, rabbit, partridge, *Lava*, peacock, *Bhṛngarāja* bird, deer, duck, parrot, drum sticks (*Śobhāñjana*), *Paṭola*, *Kāravellaka*, *Karkaṭa*, palm fruit, *Vyāghrī* fruit, *Udumbara* fruit, garlic pearls, fresh banana fruits, leaves of *Pattūra*, *Mūṣākarṇī* and *Arka*, *Guḍūcī*, *Triphalā* herbs, *Kapittha*, *Jambū*, *Kaśeru*, *Kamala*, *Kamala Kanda*, *Kamala Utpala*, *Kharajūra*, *Tṛikaṭu* herbs, *Tindika Phala*, *Khadira* and water melon. Riding elephants and horse is also helpful. So are walking excessively, wandering under the sun, exercising and wrestling. (B.R. 37/244-249)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

1 Avoid the etiological factors

सर्व एव च परिहरेयुः सौबीरकतुषोदकशुक्तमैरेयसुरासवतोयपयस्तैलघृतेक्षुविकारदधिपिष्टान्नाम्लयवागूपानकानि ग्राम्यानूपौदकमांसानि चेति ॥ *S.S.Ci. 11/15*

All varieties of *Prameha* patients should avoid the use of *Sauvīraka, Tuṣodaka, Śukta, Maireya, Sūra, Āsava, Water, Milk, Oil, Ghee,* products of sugarcane juice, curd, food prepared from flour- *Yavāgu, Pānaka,* which are sour, and meat of domestic and aquatic animals.

Prohibitions: The patient suffering from *Prameha*, should avoid suppression of urine urge, smoking, fomentation and bloodletting therapies, remaining seated by side of the soft cushions, sleeping during the day hours and indulging in intercourse. He should also not take curd in large quantity, fresh cereals, date fruits, incomptiable food items, ash gourd, sugar cane juice, contaminated water, salty, oily pastries, laxative stuff and meat of the animals from aquatic regions. (B.R. 37/250-253)

The patients suffering from the carbuncle or *Prameha Pīḍaka* should avoid the following: heavy meals, the meals that block the *Srotas* (the body fluids), bitter and hot eatables, onions and garlic, fishes and liquor. He should also avoid indulgence in excessive intercourse, exposure to fire and sun, hard exercises, remaining awake till late in nights and the like. (B.R. 38/28-29).

MCQs

1. *Picchilaguna* predominant *Prameha* is

a. *Ikshu* b. *Shukra*

c. *Shukla* d. *Alala*

2. *Shveta-Snigdhaguna* predominant *Prameha* is

a. *Ikshu* b. *Shukra*

c. *Shukla* d. *Aalala*

3. *Katurasa* predominant *Prameha* is

a. *Neela* b. *Haridra*

c. *Amla* d. *Manjishta*

4. *Amlarasa* predominant *Prameha* is

a. *Neela* b. *Haaridra*

c. *Kaala* d. *Manjishta*

5. *Ushnaguna* predominant *Prameha* is

a. *Neela* b. *Haaridra*

c. *Kala* d. *Manjishta*

6. Vatikapramehaupadrava

a. 12 b. 7

c. 10 d. 16

7. *Visragandha* predominant *Prameha* is

a. *Neela* b. *Haaridra*

c. *Amla* d. *Manjishta*

8. *Avegi* - Clinical feature of *Prameha* is seen in

a. *Shanairmeha* b. *Hastimeha*

c. *Madhu-Meha* d. All *Prameha*

9. *Mutra-kricchratva* - Clinical feature of *Prameha* is seen in

a. *Shanairmeha* b. *Hastimeha*

c. *Madhu-Meha* d. All *Prameha*

10. *Dushya* in *Hasti-Meha* is

a. *Rasa* b. *Rakta*

c. *Lasika* d. All *Kledas*

11.*Rasa* of *mutra* in Madhu-*Meha* is

a. *Madhura* b. *Lavana-Madhura*

c. *Kashaya-Madhura* d. *Tikta-Madhura*

12.*Rasa* of *mutra* in *Prameha-mukta* is

a. *Madhura* b. *Madhura-Lavana*

c. *Kashaya-Madhura* d. *Tikta-Katu*

13. *Sukha Sadhya Prameha* will be having

a. *Tulyadosha-dushya* b. *Tulyadosha-ritu*

c. *Tulyadosha- desha* d. None

14. *Kasa* develops as *Upadrava* of

a. *Kapha Prameha* b. *Pitta-Prameha*

c. *Vata-Prameha* d. Both a & c

15. *AvaranajanyaMadhu-Meha* is

a. *Sukhasadhya* b. *Kricchrasadhya*

c. *Yapya* d. *Asadhya*

16. *Hasta-tala Daha* in *Prameha* is due to

a. *Roga-Svabhaava* b. *Dosha-Prabhaava*

c. *Vyadhi-Prabhava* d. *Vyadhi-Svabhaava*

17. *Mutramapicchila, anavila, vishada, tikta, katukatka* are the characteristic properties ofurine indicates

a. Recovery from *Prameha*

b. Recovery from *Mutraghata*

c. Recovery from *Mutrakricchra*

d. Recovery from *Ashmari*

18.*Mahavasthuparigraha* is the feature if which *Pramehapitaka*

a. Vinata *b. Kacchapika*
c. Saravika *d. Jalini*

19. Kshiprapaka maharaja is the feature of which *Pramehapitaka*

a. Sarshapika *b. Masurika*
c. Vinatha *d. Alaji*

Answer

1.	d	2.	b	3.	b
4.	a	5.	c	6.	b
7.	d	8.	b	9.	a
10.	c	11.	c	12.	d
13.	a	14.	d	15.	b
16.	c	17.	a	18.	b
19.	a				

CHAPTER

39

Diabetes Mellitus and Pancreatitis

Diabetes Mellitus

1. Diabetes mellitus is a clinical syndrome characterised by hyperglycaemia caused by absolute or relative deficiency of insulin.
2. Hyperglycaemia has many causes but is most commonly due to type I or type 2 diabetes.
3. Lack of insulin affects the metabolism of carbohydrate, protein and fat, and can cause significant disturbance of water and electrolyte homeostasis
4. Death may result from acute metabolic complication
5. Longstanding metabolic derangement is associated with functional and structural changes in many organs, particularly those of the vascular system, which lead to the clinical 'complications' of diabetes. These characteristically affect the eyes, the kidney and the nervous system.
6. Hyperglycaemia represents an independent risk factor for disease of both small and large blood vessels.
7. The incidences of both type 1 and type 2 diabetes are rising; it is estimated that, in the year 2000, 171 million people had diabetes, and this is expected to double by 2030.
8. This global pandemic principally involves type 2 diabetes and is associated with greater longevity, obesity, unsatisfactory diet, sedentary lifestyle and increasing urbanisation.
9. Many cases of type 2 diabetes remain undetected.
10. The prevalence of both types of diabetes varies considerably around the world, and is related to differences in genetic and environmental factors.

Comparative features of Type1 and Type 2 Diabetes

	Type 1	Type 2
Typical age at onset	< 40yrs	> 50yrs
Duration of symptoms	Weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Autoantibodies	Yes	No
Diabetic complications atdiagnosis	No	25%
Family history of diabetes	Uncommon	Common
Other autoimmune disease	Common	Uncommon

Aetiology and pathogenesis of diabetes mellitus

1. In both of the common types of diabetes, environmental factor interact with genetic susceptibility to determine which people develop the clinical syndrome and the timing of its onset.
2. However, the underlying genes, precipitating environmental factors and pathophysiology differ substantially between Type 1 and Type 2 diabetes.
3. Type 1 diabetes was previously termed 'insulin-dependent diabetes mellitus' (IDDM) and, is invariably associated with profound insulin deficiency requiring replacement therapy.
4. Type 2 diabetes was previously termed 'non-insulin-dependent diabetes mellitus' (NIDDM) because patients retain the capacity to secrete some insulin but exhibit impaired sensitivity

to insulin (insulin resistance) and initially can usually be treated without insulin replacement therapy.

5. However, 20% or more of patients with type 2 diabetes will ultimately develop profound insulin deficiency requiring replacement therapy so that IDDM and NIDDM were misnomers.

Type 1 diabetes mellitus Pathology

Type 1 diabetes is a T cell-mediated autoimmune disease

↓

involving destruction of the insulin-secreting b cells in the pancreatic islets which takes place over many years.

↓

Hyperglycaemia accompanied by the classical symptoms of diabetes occurs only when 70-90% of b cells have been destroyed.

↓

'Insulitis' infiltration of the islets with mononuclear cells containing activated macrophages, helper cytotoxic and suppressor T lymphocytes, natural killer cells and B lymphocytes

↓

Type 1 diabetes mellitus

↓

Hyperglycaemia

↓

Leads to glycosuria and dehydration

↓

Which in turn induces secondary hyperaldosteronism

↓

Unrestrained lipolysis and proteolysis result in weight loss, increased gluconeogenesis and ketogenesis.

↓

Ketoacidosis occurs when generation of ketone bodies exceeds the capacity for their metabolism.

↓

Elevated blood H+ ions drive K+ out of the intracellular compartment, while secondary hyperaldosteronism encourages urinary loss of K+.

↓

Hyperglycaemic symptoms (thirst, polyuria, nocturia and fatigue), infections and weight loss

↓

May have developed ketoacidosis

Actiology

A. Genetic predisposition

1. Genetic factors account for about one-third of the susceptibility to type 1 diabetes, the inheritance of which is polygenic
2. Over 20 different regions of the human genome show some linkage with type 1 diabetes but most interest has focused on the human leucocyte antigen (HLA) region within the major histocompatibility complex on the short arm of chromosome 6; this locus is designated IDDM 1.

B. Environmental factors : Although genetic susceptibility appears to be a prerequisite for type 1 diabetes, the concordance rate between monozygotic twins is less than 40% and environmental factors have an important role in promoting clinical expression of the disease. Although hypotheses around, the nature of these environmental factors is uncertain.

Clinical features

1. Thirst,
2. Polyuria
3. Nocturia
4. Fatigue
5. Infections
6. Weight loss
7. Ketoacidosis as a complication

Type 2 diabetes mellitus Pathology

Type 2 diabetes is a more complex condition

↓

Because there is a combination of resistance to the actions of insulin in liver and muscle together with impaired pancreatic b cell function

↓

Leading to 'relative' insulin deficiency.

↓

Insulin resistance appears to come first,

↓

Leads to elevated insulin secretion in order to maintain normal blood glucose levels.

↓

In susceptible individuals the pancreatic b cells are unable to sustain the increased demand for insulin

↓

A slowly progressive insulin deficiency develops.

↓

Type 2 diabetes mellitus

↓

Intercurrent illness, e.g. with infections

↓

increases the production of counter-regulatory hormones such as cortisol,
growth hormone and catecholamines.

↓

This can precipitate an acute exacerbation of insulin resistance and insulin deficiency.

↓

Result in more severe hyperglycaemia and

↓

Hyperosmolar nonketotic coma

Risk factors

1. Central (visceral) obesity
2. Hypertension
3. Dyslipidaemia (characterised by elevated levels of small dense low-density lipoprotein (LDL) cholesterol and triglycerides, and a low level of high-density lipoprotein (HDL) cholesterol)

Aetiology

A. Genetic predisposition :

1. Genetic factors are important in type 2 diabetes, as shown by marked differences in susceptibility in different ethnic groups and by studies in monozygotic twins where concordance rates for type 2 diabetes approach 100%.
2. Many genes are involved and the chance of developing diabetes is also influenced very powerfully by environmental factors
3. Genome-wide association studies have identified over 20 genes or gene regions that are associated with type 2 diabetes, each exerting a small effect. The largest effect is seen with variation in *TCF7L2.*

B. Environmental and other risk factors

Diet and obesity :

1. Type 2 diabetes is associated with overeating, especially when combined with obesity and underactivity.
2. Middle-aged people with diabetes eat significantly more and are fatter and less active than their non-diabetic siblings.
3. The risk of developing type 2 diabetes increases tenfold in people with a body mass index (BMI) $>$30kg/m2
4. Obesity probably acts as a diabetogenic factor (through increasing resistance to the action of insulin) only in those who are genetically predisposed both to insulin resistance and to b cell failure.
5. Sweet foods rich in refined carbohydrate consumed frequently may increase the demand for insulin secretion, while high-fat foods may increase FFAs and exacerbate insulin resistance.

Age

Type 2 diabetes is more common in the middle-aged and elderly

Clinical features of Type 2 DM/ hyperglycaemia :

- Thirst, dry mouth
- Polyuria
- Nocturia
- Tiredness, fatigue, lethargy
- Noticeable change in weight (usually weight loss)
- Blurring of vision
- Pruritus vulvae, balanitis (genital candidiasis)
- Nausea; headache
- Hyperphagia; predilection for sweet foods
- Mood change, irritability, difficulty in concentrating, apathy

Pregnancy

The term 'gestational diabetes' refers to hyperglycaemia occurring for the first time during pregnancy. Repeated pregnancy increases the likelihood of developing irreversible diabetes, particularly in obese women; 80% of women with gestational diabetes ultimately develop permanent diabetes.

Other forms of diabetes

1. Some acquired disorders, notably other endocrine diseases such as acromegaly or Cushing's syndrome, can precipitate type 2 diabetes in susceptible individuals.
2. A number of unusual genetic diseases are associated with diabetes.
3. In rare families, diabetes is caused by single gene defects with autosomal dominant inheritance. These subtypes constitute less than 5% of all cases of diabetes and typically present as'maturity-onset diabetes of the young' (MODY), i.e. non-insulin-requiring diabetes presenting before the age of 25 years .

Aetiological classification of diabetes mellitus

A. Type 1 diabetes

- Immune-mediated
- Idiopathic

B. Type 2 diabetes

C. Other specific types

- Genetic defects of b cell function
- Genetic defects of insulin action (e.g. leprechaunism, lipodystrophies)
- Pancreatic disease (e.g. pancreatitis, pancreatectomy, neoplastic disease, cystic fibrosis, haemochromatosis, fibrocalculous pancreatopathy)
- Excess endogenous production of hormonal antagonists to insulin (e.g. growth hormone-acromegaly; glucocorticoids-Cushing's syndrome; glucagonglu-cagonoma; catecho-lamines-phaeochromocytoma; thyroid hormones-thyroto-xicosis)
- Drug-induced (e.g. corticosteroids, thiazide diuretics, phenytoin)
- Viral infections (e.g. congenital rubella, mumps, Coxsackie virus B)
- Uncommon forms of immune-mediated diabetes
- Associated with genetic syndromes (e.g. Down's syndrome; Klinefelter's syndrome; Turner's syndrome; DIDMOAD (Wolfram's syndrome) -diabetes insipidus, diabetes mellitus, optic atrophy, nerve deafness; Friedreich's ataxia; myotonic dystrophy)

D. Gestational diabetes

Investigations

I. Urine testing

A. Glucose

1. Testing the urine for glucose with dipsticks is a common screening procedure for detecting diabetes.
2. If possible, testing should be performed on urine passed 1-2 hours after a meal to maximise sensitivity.
3. Glycosuria always warrants further assessment by blood testing

B. Ketones

1. Ketone bodies can be identified by the nitroprusside reaction, which measures acetoacetate, using either tablets or dipsticks.
2. Ketonuria is therefore not pathognomonic of diabetes but, if associated with glycosuria, the diagnosis of diabetes is highly likely.
3. In diabetic ketoacidosis, ketones can also be detected in plasma using dipsticks.

C. Protein

1. Microalbuminuria or proteinuria, in the absence of urinary tract infection, is an important indicator of the development of diabetic nephropathy and/or increased risk of macrovascular disease

II. Blood testing

Glucose-

1. Glucose concentrations are lower in venous than in arterial or capillary (fingerprick) blood.
2. Whole blood glucose concentrations are lower

than plasma concentrations because red blood cells contain relatively little glucose.

3. In general, venous plasma values are the most reliable for diagnostic purposes

   Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL)

   Random plasma glucose ≥ 11.1 mmol/L (200mg/dL)

Indications for oral glucose tolerance test

- Fasting plasma glucose 6.1-7.0mmol/L (110-126mg/dL)
- Random plasma glucose 7.8-11.0 mmol/L (140-198 mg/dL)

How to perform an oral glucose tolerance test (OGTT) Preparation before the test

- Unrestricted carbohydrate diet for 3 days
- Fasted overnight for at least 8 hrs
- Rest for 30 mins
- Remain seated for the duration of the test, with no smoking

Sampling

Plasma glucose is measured before and 2 hrs after a 75 g oral glucose drink

Interpretation (venous plasma glucose)

	Fasting	2 hrs after glucose load
Fasting	6.1-6.9mmol/L	< 7.8mmol/L
Hyperglycaemia	(110-125mg/dL)	(< 140mg/dL)
Impaired glucose	< 7.0 mmol/L	7.8-11.0 mmol/L
tolerance	(< 126 mg/dL)	(140-199 mg/dL)
Diabetes	≥ 7.0 mmoi/L	≥ 11.1 mmol/L
	(≥ 126 mg/dL)	(≥ 200 mg/dL)

Diagnosis of diabetes

- Patient complains of symptoms suggesting diabetes :
- Test urine for glucose and ketones
- Measure random or fasting venous blood glucose. Diagnosis confirmed by':

  Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL)

  Random plasma glucose ≥ 11.1 mmol/L (200mg/dL)

  Indications for oral glucose tolerance test (see Box 21.10)

- Fasting plasma glucose 6.1-7.0mmol/L (110-126mg/dL)
- Random plasma glucose 7.8-11.0 mmol/L (140-198 mg/dL

III. Glycated haemoglobin

1. Glycated haemoglobin provides an accurate and objective measure of glycaemic control over a period of weeks to months.
2. This allows assessment of glycaemic control by repeated measurements every few months in patients with known diabetes, but current assays may not be sufficiently sensitive to make a diagnosis of diabetes and are usually within the normal range in patients with impaired glucose tolerance.
3. Although HbAlc concentration reflects the integrated blood glucose control over the lifespan of the erythrocyte (120 days), half of the erythrocytes are replaced in 60 days and HbAlc is most sensitive to changes in glycaemic control occurring in the month before measurement.

Acute Pancreatitis

- Pancreatitis is an inflammatory process in which pancreatic enzymes autodigest the gland.
- The gland sometimes heals without any impairment of function or any morphologic changes; this process is known as acute pancreatitis.
- Pancreatitis can also recur intermittently, contributing to the functional and morphologic loss of the gland; recurrent attacks are referred to as chronic pancreatitis.

Etiology

Biliary tract disease& Alcohol

- Long-standing alcohol consumption and biliary stone disease cause most cases of acute pancreatitis, but numerous other etiologies are known.

- As many as 70% of cases of idiopathic pancreatitis are secondary to biliary microlithiasis.

Endoscopic retrograde cholangiopancreatography

Pancreatitis occurring after endoscopic retrograde cholangiopancreatography (ERCP) is probably the third most common type (accounting for approximately 4% of cases).

Trauma

Abdominal trauma (approximately 1.5%) causes an elevation of amylase and lipase levels in 17% of cases and clinical pancreatitis in 5% of cases.

Drugs

Drugs definitely associated with acute pancreatitis include the following:

- Azathioprine
- Sulindac
- Valproic acid,
- Methyldopa
- Furosemide
- Sulfonamides
- Tetracycline
- Didanosine
- Estrogens
- 6-Mercaptopurine
- Pentamidine
- 5-aminosalicylic acid compounds
- Corticosteroids
- Octreotide

Drugs probably associated with acute pancreatitis include the following:

- Chlorothiazide and hydrochlorothiazide
- Methandrostenolone (methandienone)
- Metronidazole
- Phenformin
- Procainamide
- Nitrofurantoin
- Piroxicam
- Colaspase
- Chlorthalidone
- Combination cancer chemotherapy drugs (especially asparaginase)
- Cimetidine
- Cisplatin
- Cytosine arabinoside
- Diphenoxylate
- Ethacrynic acid

Clinical presentation -Signs and symptoms

Symptoms of acute pancreatitis include the following:

- Abdominal pain (cardinal symptom): Characteristically dull, boring, and steady; usually sudden in onset and gradually becoming more severe until reaching a constant ache; most often located in the upper abdomen and may radiate directly through to the back
- Nausea and vomiting, sometimes with anorexia
- Diarrhea

Patients may have a history of the following:

- Recent operative or other invasive procedures
- Family history of hypertriglyceridemia
- Previous biliary colic and binge alcohol consumption (major causes of acute pancreatitis)

The following physical findings may be noted, varying with the severity of the disease:

- Fever (76%) and tachycardia (65%); hypotension
- Abdominal tenderness, muscular guarding (68%), and distention (65%); diminished or absent bowel sounds
- Jaundice (28%)
- Dyspnea (10%); tachypnea; basilar rales, especially in the left lung
- In severe cases, hemodynamic instability (10%) and hematemesis or melena (5%); pale, diaphoretic, and listless appearance
- Occasionally, extremity muscular spasms secondary to hypocalcemia

The following uncommon physical findings are associated with severe necrotizing pancreatitis:

- Cullen sign (bluish discoloration around the umbilicus resulting from hemoperitoneum)
- Grey-Turner sign (reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes); more

commonly, patients may have a ruddy erythema in the flanks secondary to extravasated pancreatic exudate

- Erythematous skin nodules, usually no larger than 1 cm and typically located on extensor skin surfaces; polyarthritis

Ancillary investigations symptoms and signs

Laboratory tests are performed to support the clinical impression, such as the following:

- Serum amylase and lipase
- Liver-associated enzymes
- Blood urea nitrogen (BUN), creatinine, and electrolytes
- Blood glucose
- Serum cholesterol and triglyceride
- Complete blood count (CBC) and hematocrit; Neutrophil-Lymphocyte Ratio (NLR)
- C-reactive protein (CRP)
- Arterial blood gas values
- Serum lactic dehydrogenase (LDH) and bicarbonate
- Immunoglobulin G4 (IgG4): elevated in autoimmune pancreatitis

Diagnostic imaging Modalities employed include the following:

- Abdominal radiography (limited role): Kidneys-ureters-bladder (KUB) radiography with the patient upright is primarily performed to detect free air in the abdomen
- Abdominal ultrasonography (most useful initial test in determining the etiology, and is the technique of choice for detecting gallstones)
- Endoscopic ultrasonography (EUS) (used mainly for detection of microlithiasis and periampullary lesions not easily revealed by other methods)
- Abdominal computed tomography (CT) scanning (generally not indicated for patients with mild pancreatitis but always indicated for those with severe acute pancreatitis)
- Endoscopic retrograde cholangiopancreatography (ERCP); to be used with extreme caution in this disease and never as a first-line diagnostic tool
- Magnetic resonance cholangiopancreatography (MRCP) (not as sensitive as ERCP but safer and noninvasive)

Other diagnostic modalities include the following:

- CT-guided or EUS-guided aspiration and drainage
- Genetic testing

Chronic Pancreatitis

- Chronic pancreatitis is commonly defined as a continuing, chronic, inflammatory process of the pancreas, characterized by irreversible morphologic changes.

Signs and symptoms

- For most patients with chronic pancreatitis, abdominal pain is the presenting symptom. The patient experiences intermittent attacks of severe pain, often in the mid-abdomen or left upper abdomen and occasionally radiating in a bandlike fashion or localized to the midback. The pain may occur either after meals or independently of meals, but it is not fleeting or transient and tends to last at least several hours.
- Other symptoms associated with chronic pancreatitis include diarrhea and weight loss.

MCQs

1. Which criteria is useful for the diagnosis of Diabetes mellitus (DM)

a. An fasting plasma glucose (FPG), 7.0 mmol/L (126 mg/dL), a glucose & gt;11. 1mmol/L (200 mg/dL) 2 h after an oral glucose challenge, or an A1C 6.5% .

b. A random plasma glucose concentration 11.1 mmol/L (200 mg/dL)accompanied by classic symptoms of DM (polyuria, polydipsia, weight loss)

c. both

d. Symptoms of diabetes plus a glucose &gt; 11.1mmol/L (200 mg/dL) 2 h after anoral glucose challenge.

2. Venous blood glucose is about —— less than capillary blood due to tissue utilization
   a. 9 mg/dl b. 10 mg/dl
   c. 7 mg/dl d. 11 mg/dl

3. A faint blue discoloration around the umbilicus (Cullen&#39;s sign) may occur as the result ofhemoperitoneum, and a blue-red-purple or green-brown discoloration of the flanks (Turner's sign)reflects tissue catabolism of haemoglobin indicates ————
   a. Mild Acute Pancreatitis
   b. Severe Acute Pancreatitis
   c. Chronic Pancreatitis
   d. Severe necrotizing pancreatitis

Answer

1.	c	2.	c	3.	d

CHAPTER 40

Vatavyadhi

Introduction

Excessive indulgence in eatables which are dry, cold, scanty, quickly digestible; sexual intercourse, keeping awake at nights, improper treatments, excessive discharge of *doṣa* and blood from the body due to administration of inappropriate (*pañcakarma* and other) therapy, fasting, swimming, long distance walking and such other exercises; loss or depletion of body tissues, worry, grief, extreme debility due to protracted diseases, habit of suppression of natural urges of the body, production of *āma* (undigested intermediary metabolities), trauma, fasting or diseases of vital organs, riding fast on elephants, starvation, injury or camel, horse or falling during such riding and the like, generate the channels (of *vāta*) in the body to become empty or dry which in turn aggravate *vāyu* which, afflicting the patent channels of the body, gives rise to various generalised diseases or to those localised to an part of the body.

Importance of Vātavyādhi

स्वयम्भूरेष भगवान् वायुरित्यभिशब्दितः ॥
स्वातन्त्र्यान्नित्यभावाच्च सर्वगत्वात्तथैव च ।
सर्वेषामेव सर्वात्मा सर्वलोकनमस्कृतः ॥
स्थित्युत्पत्तिविनाशेषु भूतानामेष कारणम् ।
अव्यक्तो व्यक्तकर्मा च रूक्षः शीतो लघुः खरः ॥
तिर्यग्गो द्विगुणश्चैव रजोबहुल एव च ।
अचिन्त्यवीर्यो दोषाणां नेता रोगसमूहराट् ॥
आशुकारी मुहुश्चारी पक्वाधानगुदालयः ।

S.S.Ni. 1/5-9

Vāta is known as self born and omnipotent because of its dependence, eternity, and all pervasiveness; among all entities, this has universal characters and is worshipped by the entire world because this is the cause of origin, sustenance and destruction of living being, this is unmanifest, but manifest by actions, non unctuous, cold, light, rough, moving obliquely having two properties (sound & touch), with predominance of *Rajas*, having incomprehensible powers, carrier of *Doṣās* & *Dūṣyās,* distinguished in congregation of diseases, quick acting, frequently moving and situated in *Pakvāśaya* and *Guda.*

दोषधात्वग्निसमतां संप्राति विषयेपु च ।
क्रियाणामानुलोम्यं च करोत्यकुपितोऽनिलः ॥

S.S.Ni. 1/10

Equilibrium state of *Doṣās, Dhātus* and *Agni* is dependent on *Vāta.* It also receives the sense objects and induces actions related to physical, speech and mind.

वायोरतिबलत्वेनाशुकारित्वेन च गरीयसत्वात्तद्विकाराणां दुःसाध्यत्वादाश्चेवात्ययकरत्वाद्विशिष्टचिकित्सात्वाद्वात-व्याध्यभिधानाम् । *M.Ni. 22/1 (MK)*

वातेन जनितो व्याधिर्वातव्याधिः । *M.Ni. 22/1 (MK)*

वातजनितोऽसाधारण व्याधिर्वातव्याधिरिति ।

M.Ni. 22/1 (MK)

Disturbed and pain causing *Vāta* is responsible for the development of *Vātavyādhi.* Uncommon disease caused by *Vāta* is *Vātavyādhi. Vātavyādhis* are said to be critical because *Vāta* being the most powerful of all the *Doṣās,* having the capacity to manifest acute diseases, nature of the disease is severe, these diseases are difficult for management, needs emergency treatment and special therapeutic has to be designed.

शकत्यानु प्रासशत्याऽथ ज्वरादौ प्रतिपाद्यते ।
सर्वव्याधिनिदानं वा वातव्याधिनिरुपणम् ॥

(वातव्याधि निदान) *(Y.R. 400p)*

Vāta being independent manifest *Jvarādi* diseases by its own or in combination with *Doṣa, Dhātu & Mala*; or *Vātavyadhi* is the root cause for the manifestation of almost all diseases.

गतिः कालकृता चैषा चयाद्या पुनरुच्यते ।
गतिश्च द्विविधा दृष्टा प्राकृति वैकृति च या ॥
पित्तादेवोष्मणः पक्तिर्नराणामुपजायते ।
तच्च पित्तं प्रकुपित्तं विकारान् कुरुते बहून् ॥

प्राकृतस्तु बलं श्लेष्मा विकृतो मल उच्यते। स चैवौजः
स्मृतः काये स च पाप्मोपदिश्यते॥
सर्वा हि चेष्टा वातेन स प्राणः प्राणिनां स्मृतः। तेनैव
रोगा जायन्ते तेन चैवोपरुध्यते॥
C.S.Su. 17/115-118

There are two types of movement of *Doṣās* inside the body i.e. normal & abnormal. *Pitta* having the nature of heat, is responsible for digestion in living beings in normal conditions but when it becomes vitiated causes many diseases. *Kapha* in normal state, is responsible for strength, but during abnormal condition it becomes waste product. Normal *Kapha* is considered as *Ojas* while abnormal one is said to be sinful because it induces misery.

Similarly *Vāta* in its normal state is responsible for movement, activities, life supporter etc. but in case of abnormal conditions it produces disorders and stops the vital breathing.

वायुरायुर्बलं वायुर्वायुर्धाता शरीरिणाम्।
वायुर्विश्वमिदं सर्वं प्रभुर्वायुश्च कीर्तितः ॥
C.S.Ci. 28/3

Vāta is life, strength and sustainer of living beings. *Vāta* is the pervasive, and it is the controller of all activities of living beings.

अव्याहतगतिर्यस्य स्थानस्थः प्रकृतौ स्थितः । वायुः
स्यात्सोऽधिकं जीवेद्वीतरोगः समाः शतम् ॥
C.S.Ci. 28/4

Person can live longer upto 100 years without suffering from any diseases, if *Vāta* moves unimpaired, located in its own place & it is in its natural state (homeostasis).

Diseases of *Vātavyādhi* like *Āvaraṇa*, *Dhātustha Doṣaja Vikāra* etc. described in first volume of my book.

वातव्याधि निदान (*Vātavyādhi Nidāna*)

रूक्षीशीताल्पलध्वन्नव्यवायातिप्रजागरैः ।
विषमादुपचाराच्च दोषासृक्स्त्रवणादति ॥
लङ्घनप्लवनात्यध्ववसामातिविचेष्टितैः ।
धातूनां संक्षयाच्चिन्ताशोकरोगातिकर्षणात् ॥
दुःखशय्यासनात् क्रोधाद्दिवास्वप्नाद्भयादपि ।
वेगसंधारणादामादभिघातादभोजनात् ॥
मर्माघाताद्गजोष्ट्राश्वशीघ्रयानापतंसनात् ।
C.S.Ci. 28/15-18

Vāta gets aggravated by adopting following *Nidānās* : Excess consumption of ununctous, cold, scanty, & light food; excessive sexual intercourse; excess vigilance; improper therapeutic procedures; administration of therapies which cause excessive elimination of *Doṣās* and *Rakta,* excess fasting; excessive movements such as leaping, jumping, warfaring and physical exercise; depletion of *Dhātus*; excessive emaciation due to worry, grief and affliction of disease; sleeping over uneven beds & sitting in odd postures; anger; day sleep fear and suppression of natural urges; *Āma doṣa;* trauma; obstinence from food; injuries to vital body parts; falling down from elephants, horses and other fast moving vehicles.

वायोर्धातुक्षयात् कोपो मार्गस्यावरणेन च (वा)।
C.S.Ci. 28/59

Vāta gets aggravated by two different distinct causes i.e. 1) by the depletion of *dhātus* and 2) by the occlusion of channels.

वातव्याधि संप्राप्ति (*Vātavyādhi Samprāpti*)

वातपित्तकफा देहे सर्वस्त्रोतोऽनुसारिणः ॥
वायुरेव हि सूक्ष्मत्वाद्वयोस्तत्रात्युदीरणः ।
कुपितस्तौ समुद्धूय तत्र तत्र क्षिपन् गदान् ॥
करोत्यावृतमार्गत्वाद्रसादींश्चोषयेत् ।
C.S.Ci. 28/59-61

देहे स्त्रोतांसि रिक्तानि पूरयित्वाऽनिलो बली ॥
करोति विविधान् व्याधीन् सर्वाङ्गैकाङ्गसंश्रितान् ।
C.S.Ci. 28/18-19

धातुक्षयकरैर्वायुः कुप्यत्यतिनिषेवितैः ॥.
चरन् स्त्रोतःसु रिक्तेषु भृशं तान्येव पूरयन्।
तेभ्योऽन्यदोषपूर्णेभ्यः प्राप्य वाऽऽवरणं बली ॥
A.H.Ni. 15/5-6

Vāta, Pitta and *Kapha* move through all channels of circulation. Due to subtle nature of *Vāta* it impels the remaining two *Doṣās*. The aggravated *Vāta* having provoked these two *Doṣās* gets the scattered into different places of the body, & obstructs the channels of circulation leading to manifestation of various diseases and dries up the bodily tissue elements.

Above etiological factors aggravates *Vāta* in excess and excacerbated *Vāta* fills up the empty channels and moves greatly inside them or by getting enveloped by the other *Doṣās*, which have filled up the channels and produces different kinds of ailments affecting the whole body or a part of the body.

Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Doṣās* mainly *Vāta* in association with *Pitta* and *Kapha*

↓

Due to subtle nature of *Vāta* it impels the remaining two *Doṣās*

↓

The aggravated *Vāta* having provoked these two *Doṣās* gets the scattered into different places of the body

↓

Excacerbated *Vāta* fills up the empty channels and moves greatly inside them or by getting enveloped by the other *Doṣās*

↓

Which have filled up the channels and obstructs the channels of circulation and dries up the bodily tissue elements

Oleading to manifestation of various diseases

↓

Affecting the whole body or a part of the body

↓

Vātavyādhi

वातव्याधि संप्राप्ति घटक (Samprāpti Ghaṭaka of Vātavyādhi)

Doṣa : *Vāta*
Vāta → Pañca Vāta
Pitta → Pācaka
Kapha → Kledaka, Avalambaka, Tarpaka, Śleṣala
Karmataḥ Vṛddhi→Ākṣepaka, Kampa etc.
Karma Kṣaya→Pakṣāghāta, Paṅgu, Khañja, Mūkatā etc.
Dūṣya : *Dhātu→Rasādi Sapta dhātus*
Upadhātu → Sirā, Kaṇḍarā, Snāyu, Sandhi, Tvacā
Śārīrika Mala→Mūtra, Puriṣa
Dhātumala→Pitta, Kapha
Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi : *Viṣamāgni*
Āma : *Āgnijanya*
Srotas : *Vātavaha, Rasādi Dhātuvaha Srotas*
Srotoduṣṭi : *Saṅga, Vimārgagamana*
Sancārasthāna : *Sarva Śarīra*
Vyaktasthāna : *Sarvāṅga, Ekāṅga*
Udbhavasthāna : *Pakvāśayottha*
Adhiṣṭhāna : *Sarva Śarira or Part of the body/organs*
Svabhāva : *Āśukāri, Cirakāri*
Prabhāva : *Kaṣṭa Sādya*
Roga Mārga : *Madhyama*

वातव्याधि पूर्वरूप (*Vātavyādhi Pūrvarupa*)

अव्यक्तं लक्षणं तेषां पूर्वरूपमिति स्मृतम्।
आत्मरूपं तु तद्व्यक्तमपायो लघुता पुनः।

C.S.Ci. 28/19-20

Premonitory signs and symptoms are indistinct. When the signs and symptoms are manifested distinctly they are called the actual signs & symptoms, while diminution of these symptoms & signs indicates the subsidence of the disorder.

वातव्याधि रूप (*Vātavyādhi Rupa*)

Signs & symptoms of *Vātavyādhi*

सङ्कोचः पर्वणां स्तम्भो भेदोऽस्थ्नां पर्वणामपि ॥
लोमहर्षः प्रलापश्च पाणिपृष्ठशिरोग्रहः।
खाञ्ज्यपाङ्गुल्यकुब्जत्वं शोषोऽङ्गानामनिद्रता ॥
गर्भशुक्ररजोनाशः स्पन्दनं गात्रसुप्तता।
शिरोनासाक्षिजत्रूणां ग्रीवायाश्चापि हुण्डनम् ॥
भेदस्तोदार्तिराक्षेपो मोहश्चायास एव च।
एवंविधानि रूपाणि करोति कुपितोऽनिलः ॥
हेतुस्थानविशेषाच्च भवेद्रोगविशेषकृत्।

C.S.Ci. 28/20-24

The following signs & symptoms manifest in *Vātavyādhi* are as follows : Contraction, stiffness of joints; pain in bones & joints; horripilation; delirium, spasticity of hands, back & head; lameness of hands & feet; hunch back; atrophy of limbs; insomnia, destruction of foetus, semen & menses; twitching sensation and numbness in the body; crookedness of head, nose, eye, clavicular region and back; splitting pain; pricking pain; distress; convulsion; unconsciousness; exhaustion and other features as per involvement of lesions.

उपशय/पथ्य (Upaśaya/Pathya)

Auṣadha : *Nirguṇḍī taila, Mūlaka taila, Sahacara taila, Śvadaṃṣṭrādya taila, Balā taila, Amṛtādya taila, Rāsnā taila, Mūlakādya taila, Vṛṣamūlādi taila, Agurvādya taila, Aṇutaila, Sahasrapāka taila.*

Patra lavaṇa, Sneha lavaṇa (Khaṇḍa lavaṇa), Kalyāṇaka lavaṇa, Viṣagarbha taila, Mahāmāṣa taila, Mahālakṣminā-rāyaṇa taila, Ekādaśaśatika Prasāriṇi taila.

Ghṛta : *Nakulādya ghṛta, Bṛhat Chāgalādya ghṛta, Hamsādya ghṛta, Tilvaka ghṛta.*

Kvātha : *Mahāmañjiṣṭādi Kvātha, Ātmaguptādi Kvātha, Mahārāsnādi Kvātha, Māṣabalādi Kvātha*

Rasayoga : *Ardhāṅga Vātāri rasa, Amṛta Sanjivani rasāyana, Aṣṭamūrti Rasāyana, Indravāruṇi rasa, Ekāṅgavīra rasa, Kajjali, Kālakaṇṭaka rasa, Pakṣavātavidhvamsana rasa, Piṇḍi rasa, Malla sindūra, Yogendra rasa, Rasa rāja rasa, Rasādi guṭikā, Vātagajāṅkuśa rasa, Vāta rākṣasa rasa, Svacchanda bhairava rasa.*

Other medicines

Yogarāja guggula, Sanjivani Vaṭi, Trayodaśāṅga guggulu, Dvātrimśatika guggulu, Viṣa tinduka vaṭi, Kucalādi yoga, Vīrabahuṭi yoga.

अन्न (Anna) S.S.Ci. 4/13-14

Milk boiled with *Pañcamūla,* juice of sour fruits, soup of meat and corns added with more of fat (oil/ghee) is beneficial; fatty & salty foods.

विहार (Vihāra) S.S.Ci. 4/24-26

Pouring liquids on the body comfortable warm, mild massage, covering the body with thick cloth. Made up of leaves of plants like *Kumkuma, Aguru, Kuṣṭha, Elā, Tagara,* Silk, wool, hairs or cotton; residing in places with mild breeze and sunlight; use of soft bed, exposure to heat of fire and maintaining celibacy.

Diets and regimens prescribed for a patient suffering from *Vātavyādhi* diseases, in brief, are as follows: 1. Oleation (*Sneha*), fomentation (*Sveda*), massage (*Abhyaṅga*), enema (*Basti*), unctuous purgative (*Sneha Virecana*), keeping oil over the head through a cap like container made of leather (*Śirobasti*), oil massage over head (*Siraḥ Sneha*), unctuous type of smoking (*Snaihikā Dhūma*), lukewarm and unctuous gargle (*Gaṇḍuṣa*) and unctuous inhalation (*Snaihikā Nasya*) therapies; 2. Meat soup, different types of milk, meat and fat, diet prepared by adding fat, sour fruits, and unctuous as well as saline food; 3. *Kumkuma, Aguru, Patra, Kuṣṭha, Elā* and *Tagara*; 4. Heavy apparel made of milk, wool, leather and cotton; and 5. Taking bath in sun, residence in underground cellar, sleeping in soft bed, exposure to the heat of the fire and abstinence from sex. (B.R. 26/611-625)

अनुपशय/अपथ्य (*Anupaśaya/Apathya*)

Unwholesome diet and meals for the patient of *Vātavādhi* : gram, peas, *Nivāra, Veṇū, Kuruvinda* and the like varieties of paddy, flour obtained from *Kodrava* and *Śyāmā* grains, grains that are produced by grass, *Rājamāṣa*, *Mudga*, water obtained from ponds and rivers, contaminated water, barley, *Jambu*, *Kaseru*, *Tṛiṇakam*, betel nuts, *Mṛṇāla*, seeds of white beans, seed pulp of palm fruits, *Śālūka*, *Tenduka*, *Kāravellaka*, unripe palm fruits, *Śimbī*, leafy vegetables, *Udumbara*, cold water, milk of she-ass, incompatible meals, citrus liquids, dried meat, honey, citrus, sour and bitter eatables and the like.

Harmful measures and activities for the patient of *Vātavyādhi* diseases include: worrying, remaining waked upto late night, suppression of natural urges, vomiting, excess labour, *Laṅgana* (fasting), blood letting, intercourse with females, riding elephants and horse, walking a lot, lying idle for hours, bathing (particularly for the patient of paralysis), rubbing the teeth and the like. (B.R. 26/626-630).

Seronegative spondyloarthritis

It describes a group of related inflammatory joint diseases distinct from RA, which show considerable overlap in their clinical features and shared immunogenetic association with the HLA B27 antigen.

The diseases included are :

- Ankylosing spondylitis
- Reactive arthritis, including Reiter's syndrome
- Psoriatic arthritis
- Arthropathy associated with inflammatory bowel disease.

Clinical features common to seronegative spondyloarthritis

1. Asymmetrical inflammatory oligoarthritis (lower ≥ upper limb)
2. Sacroiliitis and inflammatory spondylitis
3. Inflammatory enthesitis
4. Tendency for familial aggregation
5. No association with seropositivity for RF
6. Absence of nodules and other extra-articular features of RA
7. Typical overlapping extra-articular features:
   - Mucosal inflammation: conjunctivitis, buccal ulceration, urethritis, prostatitis, bowel ulceration
   - Pustular skin lesions and nail dystrophy
   - Anterior uveitis
   - Aortic root fibrosis (aortic incompetence, conduction defects)
   - Erythema nodosum

MCQs

1. *Khudaka-Vata* is
   a. *Gridhrasi* b. *Vata-Kantaka*
   c. *Khalli* d. *Pangu*
2. *OjakritPitta* is
   a. *Sadhaka* b. *Pachaka*
   c. *Ranjaka* d. *Bhrajaka*
3. *Srotas shodhana* is done by
   a. *Prana* b. *Vyana*
   c. *Apana* d. *Samana*
4. *Srotahpreenana* is done by
   a. *Prana* b. *Udana*
   c. *Apana* d. *Samana*
5. *Rakta-Sravana* is performed by
   a. *Prana* b.*Samana*
   c. *Apana* d. *Vyana*
6. *AmbuvahasrotoVicari* is
   a. *Prana* b. *Samana*
   c. *Apana* d. *Vyanaa*
7. *Doshavaha-srotoVicari* is
   a. *Prana* b. *Vyanaa*
   c. *Apana* d. *Samana*
8. *Artavavaha-srotoVicari* is
   a. *Prana* b. *Vyanaa*
   c. *Apana* d. *Samana*
9. *Shukravaha-srotoVicari* is
   a. *Prana* b. *Vyanaa*
   c. *Apana* d. *Samana*
10. *Mano-bodhanakriya* is done by
    a. *Udana* b.*Prana*
    c. *Apana* d. *Samana*
11. *Asrigdhara* is seen in
    a. *Kaphavrtaapana* b. *Raktavrtavata*
    c. *Pittavrtaapana* d. *Pittavrtavyana*
12. *Yonou Shukrapratipatana* is done by
    a. *Prana* b. *Vyana*
    c. *Apana* d. *Samana*
13. Causes of *Vatavyadhi* is/are
    a. *Dhatukshaya* b. *Margavarana*
    c. *Abhighata* d. *Both a & b*
14. *Adhya-Vata* is
    a. *Ama-Vata* b. *Kateegraha*
    c. *Vata-Rakta*
    d. *Meda-Kaphavritavata*
15. Pavanottama
    a. *Pranavayu* b. *Udanavayu*
    c. *Vyanavayu* d. *Samanavayu*

Answer

1.	d	2.	d	3.	b
4.	d	5.	b	6.	d
7.	d	8.	d	9.	d
10.	a	11.	c	12.	b
13.	d	14.	d	15.	b

CHAPTER
41

Snayugata Vata

आक्षेपक (Ākṣepaka)

पाणिपादशिरःपृष्ठश्रोणीः स्तभ्नाति मारुतः ॥
दण्डवत्स्तब्धगात्रस्य दण्डकः सोऽनुपक्रमः ।
C.S.Ci. 28/50-51

A convulsion is a medical condition where body muscles contract and relax rapidly and repeatedly, resulting in an uncontrolled shaking of the body. A type of paroxysmal and spasmodic involuntary movements is considered in this category. Generally, convulsion is a symptom, which is observed in various clinical conditions. Excessively aggravated *vāta* causes frequent convulsions in different parts of the body drying up of the hands & legs along with blood vessels, ligaments & tendons. It is known as *Ākṣepaka.*

यदा तु धमनीः सर्वाः कुपितोऽभ्येति मारुतः ।
तदाक्षिपत्याशु मुहुमुहुर्देहं मुहुश्चरः ।
मुहुर्मुहुस्तदाक्षेपक इति स्मृतः ॥
S.S.Ni/1/50-51

Ākṣepaka is characterised by vigorous movements of the body because *vāta* pervades to all the *dhamanis.* It is called *ākṣepaka* because of the repeated occurence of the body movements (convulsions).

Pathological component

Doṣa : *Vāta pradhāna* with & without *Kapha* and *Pitta*

Dūṣya : *Rasa, rakta, sirā, dhamanī, kaṇḍarā.*

Adhiṣṭhāna : *Vātavaha nadi* and *dhamani*

Srotas : *Rasa, rakta* and *manovaha* along with *Sañjñavaha srotas*

Types of Srotoduṣṭi : *Saṅga & atipravṛtti*

Nature of disease : Difficult to be cure

Types:

पित्तश्लेष्मान्वितो वायुर्वायुरेव च केवलः ।
कुर्यादाक्षेपकं चान्यं चतुर्थमभिघातजम् ॥
(Bh.Pr. Madh.Kh. 24/168)

1. *Vātaja*
2. *Vāta-kaphaja*
3. *Vāta-pittaja* and
4. *Abhighātaja* (due to trauma)

Clinical features: Following features are generally found in *ākṣepaka*

1. The presentation is repeatedly
2. Convulsions in the body.
3. Emaciation of limbs
4. Emaciation or deformity in veins, tendons, ligament and muscles.
5. Defective extension of muscles
6. Spasm of muscles.

अपतानक (Apatānaka)

It is caused by the factors, which is described in the context of *vātavyādhi*, but abortion, excessive haemorrhage and trauma are important etilogical factors described by *Suśruta.*

Clinical features

दृष्टिं संस्तभ्य संज्ञां च हत्वा कण्ठ्येन कूजति ।
हृदि मुक्ते नरः स्वास्थ्यं याति मोहं वृते पुनः ॥
M.Ni. 22/31

सोऽपतानकसंज्ञो यः पातयत्यन्तराऽन्तरा ।
S.S.Ni 1/52

Following features are described in the text.

1. Vision becomes steady and fixed
2. Syncope/fainting attacks
3. Wheezing sound from throat
4. Remission of fits
5. Falling of persons in between the bouts of convulsion.

Sādhya-asādhyatā

गर्भपातनिमित्तश्च शोणितातिस्रवाच्च यः ।
अभिघातनिमित्तश्च न सिध्यत्यपतानकः ॥
S.S.Ni. 1/59

Suśruta has mentioned that *apatānaka* which is caused by abortion, haemorrhage and due to trauma becomes *asādhya* in nature.

दण्डाकपातनक (Daṇḍākapatānaka)

Generally it is a symptoms found in advanced stage of *Aptānaka*. Caraka saids this as *daṇḍaka*, while *Bhāva miśra* has described another disease *Kubjaka* in which there is hump at the back.

Clinical features

Excessively aggravated *Vāta* causes rigidity of the hands, legs, head, back, hips and lastly entire body becomes stiff like a stick. This condition is incurable.

पाणिपादशिरःपृष्ठश्रोणीः स्तभ्नाति मारुतः ।
दण्डवत्स्तब्धगात्रस्य दण्डकः सोऽनुपक्रमः ॥
C.S.Ci. 28/51-52

Suśruta defines that vitiated *vāta doṣa* in the process of disease, gets mixed with *āma* and *kapha doṣa* and causes the whole body stiffness like a stick.

सोऽपतानकसंज्ञो यः पातयत्यन्तराऽन्तरा ।
कफान्वितो भृशं वायुस्तास्वेव यदि तिष्ठति ॥
S.S.Ni.1/52

Sādhya-asādhyatā

It is *asādhya* in nature by *Caraka* and *Vāgbhaṭa*, while *Suśruta* proclaims that it is difficult to be cure.

अन्तरायाम (Antarāyāma)

मन्ये संश्रित्य वातोऽन्तर्यदा नाड्यौ प्रपद्यते ।
मन्यास्तम्भं तदा कुर्यादन्तरायामसंज्ञितम् ॥
अन्तरायम्यते ग्रीवा मन्या च स्तभ्यते भृशम् ।
दन्तानां दंशनं लाला पृष्ठायामः शिरोग्रहः ॥
जृम्भा वदनसङ्गश्चाप्यन्तरायामलक्षणम् ।
C.S.Ci. 28/43-45

Excessively aggravated *Vāta* inside *Manyā* (carotid regions) and afflicts the internal channels of this region causes spasticity of the neck which is called *Antarāyāma*. Neck bends inwards and the carotid region becomes exceedingly stiff associated with clenching of the teeth, salivation, contraction of the back, stiffness of the head, yawning & rigidity of the face. These are the signs & symptoms of *Antarāyāma.*

बहिरायाम (Bahirāyāma)

बहिरायाम उच्यते ॥
पृष्ठमन्याश्रिता बाह्याः शोषयित्वा सिरा बली ।
वायुः कुर्याद्धनुस्तम्भं बहिरायामसंज्ञकम् ॥
चापवन्नाम्यमानस्य पृष्ठतो नीयते शिरः ।
उर उत्क्षिप्यते मन्या स्तब्धा ग्रीवाऽवमृद्यते ॥
दन्तानां दशनं जृम्भा लालास्त्रावश्च वाग्ग्रहः ।
जातवेगो निहन्त्येष वैकल्यं वा प्रयच्छति ॥
C.S.Ci. 28/45-48

Excessively aggravated *Vāta* located in the back side of the neck causes constriction of the external *Sirās* as a result of which the body bends like a bow which is called *Bahirāyāma.* While bending backwards like a bow, the head moves towards the back, the chest gets protruded, the *Manyās* become rigid, the neck gets squeezed and the teeth become clenched along with yawning, salivation and aphasia. When the attack becomes acute, it either leads to the death of the patient or manifest serious deformity in his body.

अर्दित (Ardita)

Ācārya Mādhavakara defines *ardita* as facial paralysis in terms of - "*Ardayatyanilovaktram, arditam janayatyatah*". It means the vitiated *vāta doṣa*, affects the face and produces *ardita*. Caraka considered that it is a disorder of involvement of half face or half of the body part while *Suśruta* proclaimed that it is a disorder of only involvement of half face, which is true now a days in the light of conventional medicine.

Etiological factors, signs and symptoms, pathogenesis and prognosis of *Ardita* are as follows

गर्भिणीसूतिकाबालवृद्धक्षीणेष्वसृक्क्षये ।
उच्चैर्व्याहरतोऽत्यर्थं सादतः कङ्गिनानि च ॥
हसतो जृम्भतो भाराद् विषमाच्छयनादपि ।
शिरोनासौष्ठचिबुकललाटेक्षणसन्धिगः ॥

अर्दयित्वाऽनिलो वक्त्रमर्दितं जनयत्यतः ।
वक्रीभवति वक्त्रार्धं ग्रीवा चाप्यपवर्त्तते ॥
शिरश्चलति वाक्सङ्गो नेत्रादीनां च वैकृतम् ।
ग्रीवाचिबुकदन्तानां तस्मिन् पार्श्वे तु वेदना ॥
यस्याग्रजो रोमहर्षो वेपथुर्नेत्रमाबिलम् ।
वायुरूर्ध्वं त्वचि स्वापस्तोदो मन्याहनुग्रहः ॥
तमर्दितमिति प्राहुर्व्याधिं व्याधिविशारदाः ॥
क्षीणस्यानिमिषाक्षस्य प्रसक्तं सक्तभाषिणः ।
न सिध्यत्यर्दितं गाढं त्रिवर्षं वेपनस्य च ॥

S.S. Ni 1/68-73

The pregnant women, women after delivery, children, old persons, the debilitated, anemic persons, those who speak loudly, eating hard foods, laughing, yawning, weight lifting, improper postures in bed etc.

Due to above etiological factors *Vāta* gets aggravated and localized in places such as head, nose, lips, chin, forehead, and eyes and develops pain in these areas & cause *Ardita*.

Pathological component of Ardita

Doṣa : *Vāta* dominant *Tridoṣa*
Dūṣya : *Rasa, rakta, māṁsa, sirā, snāyu, kaṇḍarā*
Adhiṣṭhāna : half of the face
Srotas : *Rasa, rakta and vātavaha srotasa.*
Types of Srtoduṣṭi : *Saṅga, Vimārga gamana*
Status of Agni : *Viṣamāgni*
Nature of disease : In early stage- curable
In chronic stage- difficult to be cure.

अर्दित लक्षण (*Ardita Lakṣaṇa*)

Half of the face along with neck gets crooked; trembling in head; loss of speech; abnormal appearance of eye, etc., and there is twisting of neck, chin and teeth on the affected side.

अर्दित पूर्वरूप (*Ardita Pūrvarupa*)

Premonitory signs and symptoms of *Ardita* are as follows- Horripilations, tremors, turbidity of the eyes, upward movement of *Vāta*, numbness in skin, pricking pain, and stiffness in the sides of the neck & jaws.

साध्यासाध्यता (*Sādhyāsādhyatā*)

Ardita is incurable in persons who are emaciated, having fixed eyes and constantly muffled voice, which has persisted for than three years associated with shaking of the head.

पक्षाघात एवं अर्दित (*Pakṣāghāta Evam Ardita*)

अतिवृद्धः शरीरार्धमेकं वायुः प्रपद्यते ।
यदा तदोपशोष्यासृग्बाहुं पादं च जानु च ॥
तस्मिन् सङ्कोचयत्यर्धे मुखं जिह्मं करोति च ।
वक्रीकरोति नासाभ्रूललाटाक्षिहनूस्तथा ॥
ततो वक्रं व्रजत्यास्ये भोजनं वक्रदर्शिनः ।
स्तब्धं नेत्रं कथयतः क्षवथुश्च निगृह्यते ॥
दीना जिह्मा समुत्क्षिप्ता कला सज्जतिचास्य वाक् ।
दन्ताश्चलन्ति बाध्येते श्रवणौ भिद्यते स्वरः ॥
पादहस्ताक्षिजङ्घोरुशङ्खश्रवणगण्डरुक् ।
अर्धे तस्मिन्मुखार्धे वा केवले स्यात्तदर्दितम् ॥

C.S.Ci. 28/38-42

Due to consumption of *Vāta* aggravating etiological factors, *Vāta* becomes excessively excacerbated leading to afflictions half of the body and dries up the blood, hand, foot and knee and develops contracture in these parts. It causes distortion in half of the face, and curvature of the nose, eyebrows, forehead, eye and mandible. Ingested food moves tortuously to the one side of the mouth or remnants of the food; while speaking, the nose becomes curved & the eyes remains fixed; sneezing is suppressed inspite of impulse; tongue when raised becomes curved; voice becomes feeble and impeded; looseness of teeth; hearing is deficient; hoarseness of voice; pain in foot, hand, eye, calf, thigh, temple, ear & cheek. The disease may confined only to half of the body or in half of his face is known as *Ardita*.

Types of *Ardita*: Its types are not clearly mentioned in the classical texts of Ayurveda, but in *Bhāvaprakāśa* three types of *ardita* has been described such as- (Bh.Pr. madh.Kh. 24/64)

1. *Vāta* predominant
2. *Pitta* predominant
3. *Kapha* predominant

Clinical presentation

- Vitiated *vāta doṣa* produces contracture in half of the body or half of the face or both along with face and nose, eye brows, forehead, eyes and jaws also get crooked.
- There is excessive salivation and the eye on the affected side remains partially closed. Thus when the food is taken, the morsel goes on the affected side and the tongue also get affected.
- Patient may partially or completely loose his voice and there is pain in foot, hand, eyes, thigh, temple, ear and cheek.
- Besides, features like-involuntary movement of head, aphonia, immobilization of eyes, frictions of teeth, hoarseness of voice, deafness, difficulty in sneezing, anosmia, altered sensorium, fear at the time of sleep etc are described by *Vāgbhaṭa*. (C.S.Ci. 28/38-42, S.S.Ni. 1/71, A.H.Ni. 15/34-36)

Features of *Ardita* according to predominance of *Doṣas*:

1. *Vāta doṣa:* this is accompanied with excessive salivation, tremors, swelling and pain in affected lips and limbs.
2. *Pitta doṣa:* this is accompanied with fever, fainting and excessive thirst.
3. *Kapha doṣa:* there is oedema and stiffness of cheeks, neck and head. (Bh.PR. Madh.Kh. 24/65-66)

पक्षाघात (Pakṣāghāta)

Nidāna (aetiology)

Etiological factors of the disease are as follows-

1. All the etiological factors mentioned for *Vātavyādhi*
2. Bloodletting therapy

If ignorant physician perform bloodletting therapy in too hot season & after excessive sudation therapy as a result blood flows out excessively resulting in the manifestation of *Pakṣāghāta*.

अत्युष्णोऽतिस्विन्नेऽतिविद्धेऽज्ञैर्विस्रावितमतिप्रवर्त्तते ।
तदतिप्रवृत्तं शिरोऽभितापमान्ध्यमधिमन्थतिमिरप्रादुर्भावं
धातुक्षयमाक्षेपकं पक्षाघातमेकाविकारं । *S.S.Su. 14/30*

Pakṣāghāta may manifest in pregnant women, women after delivery, old people, emaciated persons & that caused by heavy bleeding and loss of sensation in the affected limbs should be refused for treatment.

गर्भिणी सूतिकाबालवृद्धक्षीणेष्वसृक्स्रुते ।
पक्षाघातं परिहरेद् वेदनारहितो यदि ॥
M.Ni. 22/43

Pakṣāghāta manifest due to injury to *Lohita marma* & *Kakṣadhara marma.*

तत्र लोहितक्षयेण मरणं पक्षाघातो वा ।
S.S.Śā. 6/25

कक्षधरे पक्षाधातः । *S.S.Śā. 6/25*

Samprāpti & Lakṣaṇa

1. Aggravated *Vāta* seizes power of half of the body i.e. on either right or left cause's dryness of *Sirā* & *Snāyu* and manifest symptoms like loss of movement, pain and loss of speech. *Vāta* causes contracture in one of the feet or hands with piercing pain & distress, is the characteristic feature of *Ekāṅga roga*. If the affliction of *Vāta* is generalised, this is known as *Sarvāṅga roga.*

2. Excessively aggravated *Vāta* inside the *Dhamani's* moves downwards, obliquely and upwards and paralyses on either side of the body i.e. right or left by loosening the bonds of the bindings of the joints is called *Pakṣāghāta* & it is characterised by loss of functions in half portions of the body, unconsciousness, falls down or dies because of being afflicted with *Vāta.*

If *Pakṣāghāta* manifest by *Vāta* alone is regarded as curable with great difficulty; if it is associated with either *Pitta* or *Kapha Doṣa* then it becomes curable and while that caused by depletion of *Dhātus* is incurable.

हत्वैकं मारुतः पक्षं दक्षिणं वाममेव वा ॥
कुर्याच्चेष्टानिवृत्तिं हि रुजं वाक्स्तम्भमेव च ।
गृहीत्वाऽर्धं शरीरस्य सिराः स्नायूर्विशोष्य च ॥
पादं संकोचयत्येकं हस्तं वा तोदशूलकृत् ।
एकाङ्गरोगं तं विद्यात् सर्वाङ्गं सर्वदेहजम् ॥
C.S.Ci. 28/53-55

पक्षाघातं भिषग्वराः ॥

यस्य कृत्स्नं शरीरार्धमकर्मण्यमचेतनम् ।
ततः पतत्यसन् वाऽपि जहात्यनिलपीडितः ॥
शुवातहतं पक्षं कृच्छ्रसाध्यतमं विदुः ।
साध्यमन्येन संसृष्टमसाध्यं क्षयहेतुकम् ॥
S.S.Ni. 1/61-63

If *Pakṣāghāta* manifest due to association with *Pitta* it manifests burning sensation, increased body temperature and fainting along with other classical features of *Pakṣāghāta*. If *Pakṣāghāta* manifest due to association with *Kapha*, it manifests clodness, swelling, heaviness along with other classical features of *Pakṣāghāta*.

दाहसन्तापमूर्च्छाः स्युर्वायौ पित्तसमन्विते ।
शैत्यशोथगुरुत्वानि तस्मिन्नेव कफान्विते ॥
M.Ni. 22/42-43

Types of Pakṣāghāta: Depending upon the body part involved, it can be devided into following three types:

1. *Ekāṅga ghāta:* Monoplegia- in this state only one hand or leg get affected.
2. *Adharāṅga ghāta:* Paraplegia- in this state both the lower extremities are affected.
3. *Sarvāṅga ghāta:* Diplegia- most of the times this is congenital defect and both arms and legs get paralyzed.

Sādhyāsādhyatā

1. *Pakṣāghāta* which has been developed only due to the vitiation of *Vāta doṣa* is *kaṣṭasādhya.*
2. If it is develop due to aggravation of *Vāta* due to loss of tissues, then it is also *kaṣṭasādhya.*
3. The one that is develop due to vitiation of *kapha & pitta* along with *Vāta* is *Sādhya.*
4. If it affects pregnant women, children, old people and debilitated persons then it is incurable.
5. If it is painless type then it is also incurable. (S.Ni. 1/63) (Bh.Pr. Madh.Kh 24/208)

In conventional system of medicine, paralysis have strong correlation with *Pakṣāghāt* of Ayurveda. It is loss of the ability to move one or more muscles. It may be associated with loss of feeling and other bodily functions. Paralysis is not usually caused by problems with the muscles themselves, but by problems with the nerves or spinal cord that the brain uses to control muscles. Therefore, a person with paralysis will usually have some form of nerve damage

कम्पवात-Kampavāta (Parkinsonisms)

Kampavāta is one of the *Vāta nānātmaja vyādhi* as explained by Caraka in *Śutrasthāna* chapter 20. It is also named as *Vepathu* by various other scholars of Ayurveda, which have similar meaning to *Kampavāta*. Features of Parkinson's disease of conventional system of medicine are comparable to the *Kampavāta* of Ayurveda. Parkinson's disease, it is also known as idiopathic or primary parkinsonism, hypo-kinetic rigid syndrome, or paralysis agitans. It is a degenerative disorder of the central nervous system. The motor symptoms of Parkinson's disease result from the death of dopamine-generating cells in the substantia nigra, a region of the midbrain; the cause of this cell death is unknown. Early in the course of the disease, the most obvious symptoms are movement-related; later, thinking and behavioral problems may arise. Other symptoms include sensory, sleep and emotional problems. The main motor symptoms are collectively called as *Parkinsonism.* Parkinson's disease is more common in older people, with most cases occurring after the age of 50.

Karapādatale kampo Dehabhramāṇadukhite I
Nidrābhango matiḥ kṣīṇā Kampavātsya lakṣalaṇam II

(Basavarājīyama)

1. Tremors in hands and feet
2. Irregular movement of body parts
3. Insomnias and
4. Reduced memory and intelligence

Conventional features of Parkinson's disease

1) Motor symptoms

- Bradykinesia- difficulty in initiating movements and slowness during executing movements

- Difficulty in getting from chair
- Hypophonia
- Tremor-Pill rolling type
- Rigidity
- Cog wheeling sensation
- Dystonia- muscle dysfunction characterized by spasms or abnormal muscle contraction.

2) **Non-motor symptoms**

- Depression & anxiety
- Cognitive impairment
- Sleep disturbance
- Sensory abnormality & Pain
- Loss of Smell
- Incontinence of urine
- Constipation

3) **Neuropsychiatric symptoms**

- Change in mood
- Altered cognition and behavior

गृध्रसी (Gṛdhrasī)

Excessively aggravated *Vāta* inside buttocks causes rigidity, pain and pinning sensation and gradually descends to low-back, knees, calves and legs. There will be repeated pulsation in these areas. *Vātakaphaja Gṛdhrasī* is characterised by drowsiness, heaviness and anorexia.

स्फिक्पूर्वा कटिपृष्ठोरुजानुजङ्घापदं क्रमात् ।
गृध्रसी स्तम्भरुक्तोदैर्गृह्णाति स्पन्दते मुहुः ॥
वाताद्वातकफात्तन्द्रागौरवारोचकान्विता ।

C.S.Ci. 28/56-57

Excessively aggravated *Vāta* inside buttocks causes rigidity, pain and pinning sensation and gradually descends to low-back, knees, calves and legs. There will be repeated pulsation in these areas.

Vātakaphaja Gṛdhrasi is characterised by drowsiness, heaviness and anorexia.

पार्ष्णिप्रत्यङ्गुलीनां तु कण्डरा याऽनिलार्दिता ।
सक्थ्नः क्षेपं निगृह्णीयाद्गृध्रसीति हि सा स्मृता ॥

S.S.Ni. 1/74

Excessively aggravated *Vāta* enters the *Kaṇḍarās* of the ankles & toes causes *Kṣepa* (pain & inability to extend) in the thighs, is known as *Gṛdharasi*.

विश्वाची गृध्रसी चोक्ता खल्ली तीव्ररुजान्विते ।

A.H.Ni. 15/55

Viśvācī and *Gṛdhrasī* when accompanied with severe pain are called *Khallī*

Pathological component

Doṣa : *Vāta* or *Vāta-kapha*

Dūṣya : *Rasa, rakta, māṁsa, meda, snāyu, kaṇḍarā* and *sirā*

Adhiṣṭhāna : *Kaṭipradeśa* and radiated towards posterior aspect of lower limb.

Srotas : *Rasa, rakta, mamsa, meda, asthi, vāta* and *sanjnavaha srotas*

Types of srotoduṣṭi : *Atipravṛtti, saṅga* and *sirāgranthi.*

Nature of disease : Acute or chronic

विश्वाची (Viśvācī)

The *Viśvācī* of Ayurveda is comparable to the brachial neuralgia or brachial neuritis of modern medicine, which is a radiating pain in the shoulder and upper arm caused by damage to a nerve in the brachial plexus of neck. It is caused by the structural nerve compression in the neck region, or it may be caused by the damage of nerves as a result of infection, inflammation, or progressive neurological disease. The pain may be sharp and stabbing and can come and go, depending on the cause. Many patients find it severe and intolerable, and usually seek treatment quickly.

Ayurveda described that excessively aggravated *Vāta* enters the *Kaṇḍarā* of the palm, fingers and back of arms causes impairement or loss of sensory and motor functions of the same. It is known as *Viśvācī*.

तलं प्रत्यङ्गुलीनां तु कण्डरा बाहुपृष्ठतः ।
बाह्वोः कर्मक्षयकरी विश्वाचीति हि सा स्मृता ॥
S.S.Ni. 1/75

Clinical features

Following are the signs and symptoms of brachial neuritis/neuralgia.

- Sudden onset of pain in shoulder, arm and neck or hand
- Weakness in arm and shoulder
- Difficulty moving arm and shoulder
- Pins and needles
- Numbness in arm
- Shoulder and hand
- Fatigue
- Malaise
- Headache
- Muscle aches and pains

Diagnosis and test

- NCV
- X-ray AP & lateral view
- MRI of cervical spine to rule out the cause of compre-ssion

खञ्ज-पंङ्गु (Khañja-Paṅgu)

Pathogenesis and clinical features

वायुः कट्यां स्थितः सक्थ्नः कण्डरामाक्षिपेद्यदा ।
तदा खञ्जो भवेज्जन्तुः पङ्गुः सक्थ्नोर्द्वयोरपि ॥
A.H.Ni. 15/45

Excessively aggravated *Vāta* in the waist invades the big tendons of the legs and then the person becomes *Khañja* (lame by one leg) and *Paṅgu,* when both legs are affected.

वायुः कट्यां स्थितः सक्थ्नः कण्डरामाक्षिपेद्यदा ।
खञ्जस्तदा भवेज्जन्तुः पङ्गुः सक्थ्नोर्द्वयोर्वधात् ॥
S.S.Ni. 1/77

Vāta gets vitiated by its own etiological factors and affects one of the important *kandara* in the leg. This causes very severe pain and constriction in the muscle. Hence the affected is unable to move properly and starts limping. This condition is called as *khanja*. When both legs are affected the person become paralysed and is called as *pangu*.

Differentiate between Gṛdhrasī and Viśwācī

S.No.	*Gṛdhrasī*	*Viśwacī*
Clinical features	Excessively aggravated *Vāta Kandaras* of the ankles & toes causes *Kṣepa* (pain & inability to extend) in the thighs, is known as Gṛdhrasī. Excessively aggravated *Vāta* inside buttocks causes rigidity, pain and pinning sensation and gradually descends to low-back, knees, calves and legs. There will be repeated pulsation in these areas.	y aggravated *Vāta* enters the *Kandara* of the palm, fingers and back of arms causes loss of functions in arms. It is known as *Viśwācī*.
Classification	1. *Vātaja Gṛdhrasī* 2. *Vātakaphaja Gṛdhrasī* is characterised by drowsiness, heaviness and anorexia.	No classification
Contemporary correlation	Sciatica	Radioulnar paralysis

Differentiate between Gṛdhrasī and Khañja

S.No.	*Gṛdhrasī*	*Khañja*
Clinical features	Excessively aggravated Vata enters the *Kandaras* of the ankles & toes causes Kṣepa (pain & inability to extend) in the thighs, is known as *Gṛdhrasī*. Excessively aggravated Vata inside buttocks causes rigidity, pain and pinning sensation and gradually descends to low-back, knees, calves and legs. There will be repeated pulsation in these areas.	Excessively aggravated vata in the waist invades either in the right or left big tendons of the legs and due to destruction in either of the thighs causes *Khañja* and makes him limp
Classification	1.*Vātaja Gṛdhrasī* 2. Vātakaphaja *Gṛdhrasī* is characterised by drowsiness, heaviness and anorexia.	No classification
Contemporary correlation	Sciatica	Monoplegia

Differentiate between Kalayakhañja and Khañja

S.No.	*Kalayakhañja*	Khañja
Clinical features	*Kalayakhañja* is characterised by shaking of the leg at the commencement of walking and limps while walking and binding of the joints will be loosened.	Excessively aggravated vata in the waist invades eithrer in the right or left big tendons of the legs and due to destruction in either of the thighs causes Khañja and makes him limp
Contemporary correlation	Lathyrism	Monoplegia/Limp

MCQs

a. व्यपेताहिक्का (Vyapeta Hikk
   a. *Apatantraka* b. *Antaraayama*
   c. *Bahyaayama* d. *Ardita*

2. *Dhanushkampa* is
   a. *Apatantraka* b. *Antaraayama*
   c. *Bahyayama* d. *Ardita*

3. *Kapothaivakujathe* is the feature of
   a. *Apatantraka* b. *Antaraayama*
   c. *Kshatajakasa* d. *Kshayajakasa*

4. *Vata* get vitiated in ________ in *Akshepaka*
   a. *Kandara* b. *Sira*
   c. *Snayu* d. *Dhamani*

5. *Jihwaathilekhana* is the *Nidana* of
   a. *Jihwasthambha* b. *Pakshaghata*
   c. *Hanusramsa* d. *Ardita*

6. Prognosis of *Sudhavatagata Pakshaghata*
   a. *Sukhasadhya* b. *Krchrasadhya*
   c. *Yapya* d. *Krichrasadhyatama*

7. *Bahucheshtapaharini* is
   a. *Visvachi* b. *Apabahuka*
   c. *Pangu* d. *Khanja*

8. Types of *Grdhrasi* as per *Caraka*
   a. 3 b. 1
   c. 2 d. 5

9. '*Ekaanga-Roga*' is

a. *Ardita* b. *Pakshaghata*

c. *Khanja* d. *Pangu*

10. When disturbed *Vayu* enters into *Snayu* causes

a. *Gridrasi* b. *Vishwachi*

c. *Apatanaka* d. *Aksepaka*

11. *Kricchrath charvana bhashanam* is the *Nidana* of

a. *Jihwasthambha* b. *Pakshaghata*

c. *Hanusramsa* d. *Ardita*

12. Site of *Samprapti* of *Jihwasthambha*

a. *Hanumoolasira* b. *Dhamani*

c. *Vakvahisira* d. *Jathrurdhwasira*

Answer

1.	d	2.	b	3.	a	4.	d	5.	c	6.	d
7.	a	8.	c	9.	b	10.	d	11.	c	12.	c

CHAPTER

42

Common Neurologic and Spine Disorders

Parkinson's disease

- Parkinson's disease is a neurodegenerative condition which affects the basal ganglia and which presents with differing combinations of slowness of movement (bradykinesia), increased tone (rigidity), tremor and loss of postural reflexes.
- Prevalence rates are similar throughout the world, though lower rates have been reported for China and West Africa.

Age : Whilst 10% of the patients are under 45 years at presentation, the incidence and prevalence both increase with age, the latter rising to over 1 % in those over 60.

Sex : Sex incidence is about equal.

Cigarette Smoke : It is less common in cigarette smokers.

Progression of disease

- If symptoms start in middle life, the disease is usually steadily progressive and likely to shorten lifespan because of the complications of immobility and tendency to fall.
- Onset after 70 is unlikely to shorten life or become severe.

Aetiology and Pathophysiology

- A small number of cases are familial in nature and mutations in several genes have now been identified as an underlying cause.
- However, in the majority the cause is unknown, and no strong genetic factors have been identified.
- The discovery that methyl-phenyl-tetrahydropyridine (MPTP) caused severe parkinsonism in young drug users suggests that the idiopathic disease might be due to an environmental toxin
- There are several features, including depletion of the pigmented dopaminergic neurons in the substantia nigra, hyaline inclusions in nigral cells (Lewy bodies), atrophic changes in the substantia nigra and depletion of neurons in the locus coeruleus.
- Reduced dopaminergic output from the substantia nigra to the globus pallidus leads to reduced inhibitory effects on the subthalamic nucleus, neurons of which become more active than usual in inhibiting activation of the cortex. This in turn results in bradykinesia.

Clinical features

- The classical syndrome of tremor, rigidity and bradykinesia may be absent initially.
- The non-specific symptoms of tiredness, aching limbs, mental slowness, depression and small handwriting (micrographia) may be noticed.
- The presentation is almost always unilateral, a resting tremor in an upper limb being a common presenting feature.
- The tremor may eventually affect the legs, mouth and tongue.
- It may remain the predominant symptom for some years.
- Bradykinesia may develop gradually.
- Most patients have difficulty with rapid fine movements, and this manifests itself as slowness of gait and difficulty with tasks such as fastening buttons, shaving or writing.
- Rigidity, or increased muscular tone, causes stiffness and a flexed posture.
- Postural righting reflexes are impaired early on in the disease, but falls tend not to occur until later.
- As the disease advances, speech becomes softer and indistinct.
- Although the features are initially unilateral, gradual bilateral involvement is the rule.
- Muscle strength and reflexes remain normal,

and plantar responses are flexor.

- There is a paucity of facial expression (hypomimia) and the blink reflex may be exaggerated and fail to habituate (glabellar tap sign).
- Eye movements are normal to standard clinical testing, provided allowance is made for the normal limitation of upward gaze with age.
- Sensation is normal and intellectual faculties are not affected initially. As the disease progresses, about one-third of patients develop cognitive impairment.

Physical abnarmalities in Parkinson's disease

General

- Expressionless face
- Greasy skin
- Soft, rapid, indistinct speech
- Flexed posture
- Impaired postural reflexes

Gait

- Slow to start walking
- Shortened stride
- Rapid, small stride length, tendency to shorten (destination)
- Reduced arm swing
- Impaired balance on turning

Tremor

Reciting (4-6 Hz) :

- Coarse, complex movements, usually first in fingers/thumb Flexion/extension of fingers

Abduction/adduction of thumb

Supination/pronation of forearm :

- May affect arms, legs, feet, jaw, tongue
- Intermittent, present at rest and when distracted
- Diminished on action

Postural (8-10 Hz) :

- Less obvious, faster, finer amplitude
- Present on action or posture, persists with movement

Rigidity :

- Cogwheel type, mostly upper limbs
- Plastic (lead pipe) type, mostly legs

Bradykinesia :

- Slowness in initiating or repeating movements
- Impaired fine movements, especially of fingers

Diagnosis and Investigations :

- The diagnosis is made clinically, as there is no diagnostic test for Parkinson's disease.
- Sometimes it is necessary to investigate patients to exclude other causes of Parkinsonism if there are any unusual features.

Tests for Wilson's diseases and imaging (CT or MRI)

- Patients presenting before the age of 50 are usually tested for Wilson's diseases, and imaging (CT or MRI) of the head may be needed if there any features suggestive of pyramidal, cerebellar or autonomic involvment, or the diagnosis is otherwise in doubt.

Stroke

1. Acute stroke is characterised by the rapid appearance (usually over minutes) of a focal deficit of brain function, most commonly a hemiplegia with or without signs of focal higher cerebral dysfunction (such as aphasia), hemisensory loss, visual field defect or brain-stem deficit.
2. Provided that there is a clear history of a rapid-onset focal deficit, the chance of the brain lesion being anything other than vascular is 5%, or less.
3. However, care needs to be taken to exclude other differential diagnoses if the symptoms progress over hours or days.
4. Confusion, memory or balance disturbance are more often due to causes other than stroke.

Several terms have been used to classify strokes, often based on the duration and evolution of symptoms

Transient Ischaemic Attack (TIA) :

1. Describes a stroke in which symptoms resolve within 24 hours-all arbitrary cutoff which has little value in practice apart from perhaps indicating that underlying cerebral

haemorrhage or extensive cerebral infarction is extremely unlikely

2. The term TIA traditionally also includes patients with transient monocular blindness (also known as amaurosis fugax), usually due to a vascular occlusion in the retina.

Clinical features

Transient symptoms, such as syncope, amnesia, confusion and dizziness, which do not reflect focal cerebral dysfunction, are often mistakenly attributed to TIA

Stroke

1. This is the term reserved for those events in which symptoms last more than 24 hours.
2. The differential diagnosis of patients with symptoms lasting a few minutes or hours is similar to those with persisting symptoms.

Progressing stroke (or stroke in evolution)

1. the focal neurological deficit worsens after the patient first presents
2. Such worsening may be due to increasing volume of infarction, haemorrhagic transformation or increasing oedema

Completed stroke

1. The focal deficit persists and is not progressing.

Aetiology

Stroke risk factors

Fixed

- Age
- Gender (male > female, except in the very young and very old)
- Race (Afro-Caribbean > Asian > European)
- Heredity
- Previous vascular event, e.g. myocardial infarction, stroke or peripheral embolism
- High fibrinogen

Modifiable

- High blood pressure
- Heart disease (atrial fibrillation, heart failure, endocarditis)
- Diabetes mellitus
- Hyperlipidaemia
- Smoking
- Excess alcohol consumption
- Polycythaemia
- Oral contraceptives
- Social deprivation

Pathophysiology

Of patients presenting with a stroke

↓

85% will have sustained a cerebral infarction due to inadequate
blood flow to part of the brain

↓

The remaining .i.e. 15% will have had an intra cerebral haemorrhage

↓

Brain imaging is required to distinguish these pathologies

↓

To guide management

↓

The combination of severe headache and vomiting at the onset of the focal neurological deficits increases the likelihood of a haemorrhagic stroke.

↓

Cerebral infarction

Aetiology

1. Cerebral infarction is mostly due to thromboembolic disease secondary to atherosclerosis in the major extra cranial arteries (carotid artery and aortic arch)
2. About 20% of infarctions are due to embolism from the heart
3. A further 20% are due to intrinsic disease of small perforating vessels (lenticulostriate arteries), producing so called 'lacunar' infarctions.

Pathophysiology

Cerebral infarction is a process which takes some hours to complete

↓

Even though the patient's deficit may be maximal close to the onset of the causative vascular occlusion

↓

After the occlusion of a cerebral artery

↓

infarction may be forestalled by the opening of anastomatic channels from other arterial territories which restore perfusion to its territory

↓

Similarly, reduction in perfusion pressure leads to compensatory homeostatic changes to maintain tissue oxygenation

↓

The changes can sometimes prevent even occlusion of a carotid artery from having any clinically apparent effect.

↓

However, if and when these homeostatic mechanisms fail

↓

the process of ischemia starts and ultimately leads to infarction unless vascular supply is restored.

↓

As the cerebral blood flow declines, different neuronal functions fail at various thresholds

↓

Once blood flow falls below the threshold for the maintenance of electrical activity

↓

Neurological deficit appear

↓

At this level of blood flow, the neurons are still viable

↓

If the blood flow increases again, function returns and the patient will have had a TIA.

↓

If the blood flow falls further

↓

A level is reached at which the process of irreversible cell death starts

↓

Hypoxia

↓

Leads to an inadequate supply of adenosine triphosphate (ATP)

↓

Which in turn leads to failure of membrane pumps, thereby allowing influx of sodium and water into the cell (cytotoxic oedema) and the release of the excitatory neurotransmitter glutamate into the extracellular fluid.

↓

Glutamate opens membrane channels, allowing the influx of calcium and more sodium into the neurons.

↓

Calcium entering the neurons activates intracellular enzymes that complete the destructive process.

↓

The release of inflammatory mediators by microglia and atrocities

↓

produces death of all cell types in the area of maximum ischemia.

↓

Cerebral infarction

Intracerebral haemorrhage

1. This usually results from rupture of a blood vessel within the brain parenchyma
2. It may also occur in patient with a subarachnoid haemorrhage if the artery rupture, into the brain substance as well as into the subarachnoid space.
3. Haemorrhage frequently occurs into an area of brain infarction.
4. If the volume of haemorrhage is large, this may be difficult to distinguish from primary intracerebral haemorrhage both clinically and radiologically.

The risk factors and underlying causes of intracerebral haemorrhage are as follows : Causes of intracerebral haemorrhage and associated risk factors :

Disease	Risk factors
Complex small vessel disease	Age
With disruption of vessel wall	Hypertension Low cholesterol
Amyloid angiopathy	Familial (rare) Age
Impaired blood clotting	Anticoagulant therapy Blood dyscrasia Thrombolytic therapy
Vascular anomaly	Arteriovenous malformation Cavernous haemangioma
Substance misuse	Alcohol Amphetamines Cocaine

Pathophysiology

The explosive entry of blood into the brain parenchyma

↓

Causes immediate cessation of function in that area

↓

As neurons are structurally disrupted and white matter fibre tracts are split apart.

↓

Thu haemorrhage itself may expand over the first minutes or hours,

↓

Or it may be associated with a rim of cerebral oedema,

↓

Which, along with the haematoma,

↓

Acts like a mass lesion to cause progression of the neurological deficit.

↓

Intracerebral haemorrhage

↓

If the enough, this can cause shift of the intracranial contents,

↓

producing transtentorial coning and sometimes rapid death

↓

If the patient survives,

↓

The haematoma is gradually absorbed,

↓

Leaving a haemosiderin -lined slit in the brain parenchyma

Clinical features

1. The neurological deficit can be identified from the patient's history and, if the deficit is persistent, from the neurological examination.
2. Features of cerebral hemisphere

   The presence of a unilateral motor deficit, a higher cerebral function deficit such as aphasia or neglect, or a visual field defect usually places the lesion in the cerebral hemisphere.
3. Features of brain stem or cerebellum

   Ataxia, diplopia and/or bilateral weakness usually indicate a lesion in the brain stem or cerebellum.
4. Reduced conscious level usually indicates a large volume lesion in the cerebral hemisphere but may result from a lesion in the brain stem or complications such as, obstructive hydrocephalus, hypoxia or severe systemic infection.

Examination of a stroke patient

Skin :

- Xanthelasma
- Rash (arteritis, splinter haemorrhages, livedo reticularis)
- Colour and temperature change (limb ischemia/deep venous thrombosis)

Eyes :

- Arcus senilis
- Diabetic changes
- Hypertensive changes
- Retinal emboli

Cardiovascular system :

- Heart rhythm (atrial fibrillation)
- Blood pressure (hypertension, hypotension)
- Jugular venous pressure (heart failure, hypovolaemia)

- Murmurs (sources of embolism)
- Peripheral pulses and bruits (generalised arteriopathy)

Respiratory system :

- Signs of pulmonary oedema
- Signs of respiratory infection

Abdomen :

- Palpable bladder (urinary retention)

Locomotor :

- Injuries sustained during collapse with stroke
- Co-morbidities which influence functional ability

Investigations

Investigation of a patient presenting with an acute stroke aims to confirm the vascular nature of the lesion, distinguish cerebral infarction from haemorrhage and identify the under lying vascular disease and risk factors

Investigation of a patient with an acute stroke

Diagnostic question	Investigation
Is it a vascular lesion?	CT/MRI
Is it ischemic or haemorrhagic?	CT/MRI
Is it a subarachnoid haemorrhage?	CT/lumbar puncture
Is there any cardiac source ofembolism?	Electrocardiogram (ECG) 24-hour ECG Echocardiogram
What is the underlying vascular disease?	Duplex ultrasound of carotids Magnetic resonance angiography (MRA) CT angiography (CTA) Contrast angiography
What are the risk factors?	Full blood count Cholesterol Blood glucose
Is there an unusual cause?	ESR Serum protein electrophoresis Clotting/thrombophilia screen

Causes and investigation of acute stroke in young patients.

Cause	Investigation
Cerebral infarct	
Cardiac embolism	Echocardiography (including transoesophageal)
Premature atherosclerosis	Serum lipids
Arterial dissection	MRI Angiography
Thrombophilia	Protein C. protein S Antithrombin III Factor V Leiden, prothrombin
Homocystinuria	Urinary amino acids Methionine loading test
Antiphospholipid antibody syndrome	Anticardiolipin antibodies/lupus anticoagulant
SLE	Antinuclear antibodies
Vasculitis	ESR CRP Antineutrophil cytoplasmic antibody (ANCA)
CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy)	MRI brain Genetic analysis Skin biopsy
Mitochondrial cytopathy	Serum lactate White cell mitochondrial DNA Muscle biopsy Mitochondrial molecular genetics
Fabry's disease	Alpha-galactosidase levels in blood
Neurovascular syphilis Primary intracerebral haemorrhage	Syphilis serology
Arteriovenous malformation (AVM)	MRI/angiography
Drug misuse	Drug screen (amphetamine, cocaine)
Coagulopathy	Prothrombin time (PT) and

	activated partial thromboplastin time (APTT) Platelet count
Subarachnoid haemorrhage	
Saccular ('berry') aneurysm	MRI/angiography
AVM	MRI/angiography
Vertebral dissection	MRI/angiography

Bell's palsy

Aetiology

1. Idiopathic facial nerve palsy or Bell's palsy is a common condition affecting all ages and both sexes.
2. The lesion is within the facial canal and may be due to reactivation of latent herpes simplex virus 1 infection.

Clinical features

1. Symptoms usually develop sub acutely over a few hours
2. Pain around the ear preceding the unilateral facial weakness.
3. Patients often describe the face as 'numb',
4. There is no objective sensory loss (except possibly to taste).
5. Hyperacusis can occur if the nerve to stapedius is involved
6. Diminished salivation and tear secretion.
7. Examination reveals an ipsilateral lower motor neuron facial nerve palsy.
8. Vesicles in the ear or on the palate indicate that the facial palsy is due to herpes zoster rather than Bell's palsy.
9. Prednisolone 40-60 mg daily for a week speeds recovery if started within 72 hours.
10. Artificial tears and ointment prevent exposure keratitis and the eye should be taped shut overnight.
11. About 80% of patients recover spontaneously within 12 weeks.
12. A slow or poor recovery is predicted by complete paralysis, older age and reduced facial motor action potential amplitude after the first week.
13. Recurrence can occur but should prompt further investigation.
14. Aberrant re-innervations may occur during recovery, producing unwanted facial movements such as eye closure when the mouth is moved, or crocodile tears' (tearing during salivation).

Motor Neuron Disorders

Motor neuron disorders (MNDs) are a clinically and pathologically heterogeneous group of neurologic diseases characterized by progressive degeneration of motor neurons; they include both sporadic and hereditary diseases.

Either or both of the following two sets of motor neurons can be affected:

- Upper motor neurons (UMNs), which originate from the primary motor cortex of the cerebrum (precentral gyrus) and possess long axons forming corticospinal and corticobulbar tracts
- Lower motor neurons (LMNs), which originate in the brainstem (cranial nerve [CN] motor nuclei) and spinal cord (anterior horn cells) and directly innervate skeletal muscles
- MNDs can be classified into those affecting primarily the UMNs, those affecting primarily the LMNs, and those affecting both, and the nomenclature is used accordingly.
- The patient's symptoms vary, depending on which set of motor neurons is involved.

The common Motor neuron disorders

- Amyotrophic lateral sclerosis (ALS)
- Primary lateral sclerosis (PLS)
- Hereditary spastic paraparesis (HSP)
- Progressive bulbar palsy (PBP), including hereditary forms
- Spinal muscular atrophy (SMA)
- X-linked spinobulbar muscular atrophy (SBMA; Kennedy disease)

- Postpolio syndrome (PPS)

Amyotrophic lateral sclerosis(ALS)

- ALS, also known as Lou Gehrig disease, is the most common neurodegenerative disease of adult onset involving the motor neuron system.
- It is a fatal disorder and is characterized by progressive skeletal muscle weakness and wasting or atrophy (ie, amyotrophy), spasticity, and fasciculations as a result of degeneration of the UMNs and LMNs, culminating in respiratory paralysis
- The etiology of sporadic amyotrophic lateral sclerosis (ALS) remains uncertain, however, ALS is considered to be a multifactorial disease that is triggered by a complex interaction of internal factors (eg, genetic susceptibility to different neuronal insults and immunologic alterations) and external factors (eg, environmental variables).
- The key theories proposed to date for the pathogenesis of ALS include glutamate-induced excitotoxicity, oxidative injury, altered mitochondrial function, cytoskeleton alterations, axonal transport dysregulation, neuroinflammation, immunomodulation, and autoimmunity.

Primary lateral sclerosis

- The etiology of primary lateral sclerosis (PLS) is unknown, but it may be similar to that proposed for ALS.
- The age of onset for primary lateral sclerosis (PLS) is usually between 40 and 60 years, with spasticity in the legs accompanied by hyperactive deep tendon reflexes, clonus, and Babinski sign.
- Patients with spastic dysarthria as an initial symptom of PLS are also described. In contrast to ALS, the LMNs remain intact, and thus, no amyotrophy is observed.

Hereditary spastic paraparesis

- More than 40 genetic loci ascribed to the different types of hereditary spastic paraparesis (HSP) have been identified; however, a clear genetic basis for most HSP types remains uncertain.
- The essential clinical findings are slowly progressive and often include severe spasticity, hyperreflexia, and weakness in a pyramidal distribution, noticeably in both lower limbs, with extensor plantar responses.

Progressive bulbar palsy

- The etiopathogenesis of Brown-Vialetto-Van Laere syndrome and Fazio-Londe disease remains elusive.
- Brown-Vialetto-Van Laere syndrome typically presents with progressive sensorineural hearing loss.

Spinal muscular atrophy

- Type I-III spinal muscular atrophy (SMA) is caused by alterations in the survival motor neuron gene (SMN).
- The clinical picture of spinal muscular atrophy (SMA) is highly variable and represents a continuum. The age of onset ranges from before birth to adulthood.

X-linked spinobulbar muscular atrophy (Kennedy disease)

- X-linked spinobulbar atrophy (SBMA) is clinically characterized by adult-onset limb and bulbar weakness, muscular atrophy, and fasciculation, with frequent occurrence of endocrine disturbances such as gynecomastia, testicular atrophy, hypercholesterolemia, and diabetes mellitus

Postpolio syndrome

- Most patients with postpolio syndrome (PPS) present with new, slowly progressive muscle weakness, frequently accompanied by muscle pain (myalgias) and fatigue, which can occur in both previously affected and unaffected muscles. This condition can also affect respiratory and bulbar muscles

Acute Transverse Myelitis (Transverse myelitis)

- Acute transverse myelitis is most commonly due to multiple sclerosis.

- It can occur in patients with vasculitis, systemic lupus erythematosus (SLE), antiphospholipid syndrome, other autoimmune disorders, mycoplasmal infections, Lyme disease, syphilis, tuberculosis (TB), COVID-19, or viral meningoencephalitis or in patients taking amphetamines, IV heroin, or antiparasitic or antifungal medications.
- Transverse myelitis occurs with optic neuritis in neuromyelitis optica (Devic disease), once considered a variant of multiple sclerosis but now considered a distinct disorder.
- The mechanism of transverse myelitis is often unknown, but some cases follow viral infection or vaccination, suggesting an autoimmune reaction.
- Inflammation tends to involve the spinal cord diffusely at one or more levels, affecting all spinal cord functions.

Clinical presentations-Symptoms and Signs

- Pain in the neck, back, or head.
- A bandlike tightness around the chest or abdomen, weakness, tingling, numbness of the feet and legs, and difficulty voiding develop over hours to a few days.
- Deficits may progress over several more days to a complete transverse sensorimotor myelopathy, causing paraplegia, loss of sensation below the lesion, urinary retention, and fecal incontinence.
- Occasionally, position and vibration sensation are spared, at least initially.
- Acute transverse myelitis occasionally recurs in patients with multiple sclerosis, SLE, or antiphospholipid syndrome.

Ancillary investigations

- MRI and cerebrospinal fluid (CSF) analysis
- Other tests to identify treatable causes
- Diagnosis of acute transverse myelitis is suggested by transverse sensorimotor myelopathy with segmental deficits.
- Guillain-Barre syndrome can be distinguished because it does not localize to a specific spinal segment.
- Diagnosis requires MRI and CSF analysis. MRI typically shows cord swelling if transverse myelitis is present and can help exclude other treatable causes of spinal cord dysfunction (eg, spinal cord compression).
- CSF usually contains monocytes, protein content is slightly increased, and IgG index is elevated (normal, = 0.85).
- A test for a marker for neuromyelitis optica IgG (NMO-IgG)
- Tests for treatable causes should include chest x-ray; PPD (purified protein derivative) for tuberculosis; serologic tests for mycoplasma, Lyme disease, COVID-19, and HIV; erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP); antinuclear antibodies; and CSF and blood Venereal Disease Research Laboratory (VDRL) tests.
- History may suggest a medication or recreational drug as a cause.
- The differential diagnosis of acute transverse myelitis includes other transverse myelopathies due to nutritional deficiencies (eg, deficiency of vitamin B12, folate, zinc, or copper), vascular insufficiency, and intraspinal tumors.

Epilepsy Organic

History of Addiction

- Alcohol withdrawal seizures and seizures because of cocaine abuse are becoming more common.
- Patients frequently lie about their use of illicit drugs or alcohol. In young adults and teenagers, a urine drug screen should be done.

History of fever

- Seizures may be induced by high fevers in children who are otherwise normal and who never develop other neurologic problems, including epilepsy. However, febrile seizures occur only in a relatively small proportion of children
- Fever should make one think of meningitis or encephalitis, or if it has been extended over a longer period of time, a cerebral abscess.

- In children, one should consider the possibility of febrile convulsions.

Are there focal neurologic signs and papilledema

- These findings are more typical of a space-occupying lesion such as a cerebral tumor, cerebral abscess, or a subdural hematoma.

Epileptogenesis

- There are a variety of conditions that have an extremely high likelihood of resulting in a chronic seizure disorder. One of the best examples of this is severe, penetrating head trauma, which is associated with up to a 45% risk of subsequent epilepsy. The high propensity for severe traumatic brain injury to lead to epilepsy suggests that the injury results in a long-lasting pathologic change in the CNS that transforms a presumably normal neural network into one that is abnormally hyperexcitable. This process is known as epileptogenesis, and the specific changes that result in a lowered seizure threshold can be considered epileptogenic factors.
- Other processes associated with epileptogenesis include stroke, infections, and abnormalities of CNS development.

Other causes

- Cerebrovascular disease
- Metabolic disorders (uremia, hepatic failure, electrolyte abnormalities, hypoglycemia, hyperglycemia)
- Alzheimer's disease and other degenerative CNS diseases

Lumbago

Aetiology

Acute lumbar disc herniation is often precipitated by trauma, usually by lifting heavy weights while the spine is flexed.

Pathophysiology

The nucleus pulposus may bulge or rupture through the annulus fibroses

↓

Giving rise to pressure on nerve endings in the spinal ligament

↓

Changes in the vertebral joints or pressure on nerve roots

↓

The altered mechanics of the lumbar spine

↓

Result in loss of lumbar lordosis

↓

And there may be spasm of the paraspinal musculature

↓

Root pressure is suggested by limitation of flexion of the hip on the affected side

↓

If the straight leg is raised (Lasegue's sign)

↓

If the third or fourth lumbar roots are involved, Lisegue's sign may be negative

↓

Pain in the back may be induced by hyperextension of the hip (femoral nerve stretch test)

↓

The roots most frequently affected are S1, L-5 and L4

Clinical features

1. The onset may be sudden or gradual.
2. Alternatively, repeated episodes of low back pain may precede sciatica by months or years.
3. Constant aching pain is felt in the lumbar region and may radiate to the buttock, thigh, calf and foot.
4. Pain is exacerbated by coughing or straining but may be relieved by lying flat.

Physical signs in lumbar root compression

Disc level	Root loss	Sensory ness	Weak- loss	Reflex
L3/L4	L4	Inner calf	Inversion of foot	Knee
L4/L5	L5	Outer calf and dorsum of foot	Dorsiflexion of hallux/toes	
L5/S1	S1	Sole and lateral foot	Plantar flexion	Ankle

Investigations

1. Plain X-rays of the lumbar spine are of little value in the diagnosis of lumbar disc disease, although they may show other conditions such as malignant infiltration of a vertebral body.
2. CT, especially using spiral scanning techniques, can provide helpful images of the disc protrusion and/or narrowing of the exit foramina.
3. MRI is the investigation of choice if available, since soft tissues are well imaged.

Lumbar Spondylosis/ osteoarthritis lumbar spine

- Typically occurs in later life and primarily involves the cervical and lumbosacral spine.

Clinical features

- Patients often complain of back pain that increases with movement and is associated with stiffness.
- The relationship between clinical symptoms and radiologic findings is usually not straightforward.
- Osteophytes or combined disk-osteophytes may cause or contribute to central spinal canal stenosis, lateral recess stenosis, or neural foraminal narrowing

Investigations

- Lumbo-sacral x-ray (ap & lateral)
- Lumbo-sacral CT
- Lumbo-sacral MRI

Lumbar Disk Disease

1. Disk disease is most likely to occur at the L4-L5 or L5-S1 levels
2. The upper lumbar levels are involved occasionally.

Aetiology

3. The cause is often unknown
4. The risk is increased in overweight individuals.
5. Genetic factors may play a role in predisposing some patients to disk disease
6. This is a common cause of chronic or recurrent low back and leg pain.
7. Disk herniation is unusual prior to age 20 years and is rare in the fibrotic disks of the elderly.

Clinical features

- The pain may be located in the low back only or referred to a leg, buttock, or hip.
- A sneeze, cough, or trivial movement may cause the nucleus pulposus to prolapse, pushing the frayed and weakened annulus posteriorly.
- With severe disk disease, the nucleus may protrude through the annulus (herniation) or become extruded to lie as a free fragment in the spinal canal.

Investigations

- A lumbar spine MRI scan or CT-myelogram is necessary to establish the location and type of pathology.
- Spine MRIs yield exquisite views of intraspinal and adjacent soft tissue anatomy.
- Bony lesions of the lateral recess or intervertebral foramen are optimally visualized by CT-myelography.
- The diagnosis of nerve root injury is most secure when the history, examination, results of imaging studies, and the EMG are concordant.
- The correlation between CT and EMG for localization of nerve root injury is between 65 and 73%.
- Up to one-third of asymptomatic adults have a lumbar disk protrusion detected by CT or MRI scans.

Sciatica Syndrome

- Sciatica is a symptom and not a diagnosis.
- Sciatica describes leg pain that is localized in the distribution of one or more lumbosacral nerve roots, typically L4-S2, with or without neurological deficit.
- The leg pain from the lumbosacral segment is termed as sciatica.

Causes

- Degenerative arthritis and disc prolapse are the common causes.

- It is often caused by a herniated intervertebral disc, compressing the L5 or S 1 ventral rami. The so-called piriformis syndrome, which may result from a variant or anomalous relationship between piriformis and the sciatic nerve, may also produce hip pain that radiates inferiorly along the course of the sciatic nerve.
- Broadly sciatica can either be because of inflammation of the sciatica nerve or because of compression of one of the extra constituting the sciatica nerve.

Inflammatory

- Sciatica neuritis
- Arachnoiditis

Nerve root compression

- Compression in the vertebral canal by disc, tumour, tuberculosis
- Compression in the intervertebral foramen due to root canal stenosis because of OA, spondylolisthesis, facet arthropathy or tumours
- Compression in the buttock or pelvis by abscess, tumour, haematoma.

Clinical features

- Sciatica is pain in the lower back and hip region, which radiates inferiorly along the posterior thigh to the leg.
- It means a pain radiating down the back of the thigh and calf.

Causes of Back or Neck Pain

Congenital/Developmental

- Spondylolysis and spondylolisthesis
- Kyphoscoliosis
- Spina bifida occulta
- Tethered spinal cord

Minor Trauma

- Strain or sprain
- Whiplash injury

Fractures

- Trauma-falls, motor vehicle accidents
- Atraumatic fractures-osteoporosis, neoplastic infiltration, exogenous steroids, osteomyelitis

Intervertebral Disk Herniation

- Degenerative
- Intervertebral foraminal narrowing
- Disk-osteophyte complex
- Internal disk disruption
- LSS with neurogenic claudication
- Uncovertebral joint disease
- Atlantoaxial joint disease (e.g., rheumatoid arthritis)
- Arthritis
- Spondylosis
- Facet or sacroiliac arthropathy
- Neoplasms-metastatic, hematologic, primary bone tumors
- Infection/Inflammation
- Vertebral osteomyelitis
- Spinal epidural abscess
- Septic disk (discitis)
- Meningitis
- Lumbar arachnoiditis
- Autoimmune [e.g., ankylosing spondylitis, reactive arthritis (formerly known as Reiter's syndrome)]
- Metabolic
- Osteoporosis-hyperparathyroidism, immobility
- Osteosclerosis (e.g., Paget's disease)
- Vascular
- Abdominal aortic aneurysm
- Vertebral artery dissection
- Other
- Referred pain from visceral disease
- Postural
- Psychiatric, malingering, chronic pain syndromes

Examination of the Back

- A physical examination that includes the abdomen and rectum is advisable.
- Back pain referred from visceral organs may be reproduced during palpation of the abdomen [pancreatitis, abdominal aortic aneurysm (AAA)] or percussion over the costovertebral angles (pyelonephritis).
- Inspection may reveal a lateral curvature of

the spine (scoliosis) or an asymmetry in the prominence of the paraspinal muscles, suggesting muscle spasm.

- Back pain of bony spine origin is often reproduced by palpation or percussion over the spinous process of the affected vertebrae.
- Forward bending is often limited by paraspinal muscle spasm; the latter may flatten the usual lumbar lordosis.
- Flexion at the hips is normal in patients with lumbar spine disease, but flexion of the lumbar spine is limited and sometimes painful.
- Lateral bending to the side opposite the injured spinal element may stretch the damaged tissues, worsen pain, and limit motion.
- Hyperextension of the spine (with the patient prone or standing) is limited when nerve root compression, facet joint pathology, or other bony spine disease is present.

Pain from Hip

- Pain from hip disease may mimic the pain of lumbar spine disease.

Patrick's sign

- Hip pain can be reproduced by internal and external rotation at the hip with the knee and hip in flexion (Patrick's sign)

Heel percussion sign

- Tapping the heel with the examiner's palm while the leg is extended (heel percussion sign).

Straight leg-raising maneuver

- With the patient supine, passive flexion of the extended leg at the hip stretches the L5 and S1 nerve roots and the sciatic nerve (straight leg-raising maneuver).
- Passive dorsiflexion of the foot during the maneuver adds to the stretch.
- While flexion to at least 80ã is normally possible without causing pain, many patients normally report a tight, stretching sensation in the hamstring muscles unrelated to back pain.
- The *straight leg-raising (SLR)* test is positive if the maneuver reproduces the patient's usual back or limb pain.
- Eliciting the SLR sign in the sitting position can help determine if the finding is reproducible.
- The patient may describe pain in the low back, buttocks, posterior thigh, or lower leg, but the *key feature is reproduction of the patient's usual pain.*

Crossed SLR sign

- The *crossed SLR sign* is positive when flexion of one leg reproduces the usual pain in the opposite leg or buttocks.
- The crossed SLR sign is less sensitive but more specific for disk herniation than the SLR sign.
- The nerve or nerve root lesion is always on the side of the pain.

Reverse SLR sign

- The *reverse SLR sign* is elicited by standing the patient next to the examination table and passively extending each leg with the knee fully extended.
- This maneuver, which stretches the L2-L4 nerve roots, lumbosacral plexus, and femoral nerve, is considered positive if the patient's usual back or limb pain is reproduced.

Neurological examination findings

- It includes a search for focal weakness or muscle atrophy, focal reflex changes, diminished sensation in the legs, or signs of spinal cord injury.

Breakaway weakness

- It is defined as fluctuating strength during muscle testing.
- It may be due to pain or a combination of pain and underlying true weakness.
- Breakaway weakness without pain is almost always due to a lack of effort.
- In uncertain cases, electromyography (EMG) can determine whether or not true weakness due to nerve tissue injury is present.
- Findings with specific nerve lumbosacral nerve root lesions are discussed below.

Lumbosacral Radiculopathy : Neurologic Features

Examination Findings

Lumbosacral Nerve Roots	Reflex	Sensory	Motor	Pain Distribution	Sign
L2	-	Upper anterior thigh	Psoas (hip flexion)	Anterior thigh	Reverse straight leg–raising sign present
L3	-	Lower anterior thigh Anterior knee	Psoas (hipflexion) Quadriceps (knee extension) Thigh adduction	Anterior thigh, knee	Reverse straight leg–raising sign present
L4	Quadriceps (knee)	Medial calf	Quadriceps (knee extension) Thigh adduction Tibialis anterior (foot dorsiflexion)	Knee, medial calf Anterolateral thigh	Reverse straight leg–raising sign present
L5	-	Dorsal surface foot Lateral calf	Peroneii (foot eversion) Tibialis anterior (foot dorsi flexion) Gluteus medius (hip abduction) Toe dorsiflexors	Lateral calf, dorsal foot, posterolateral thigh, buttocks	Straight leg–raising sign present
S1	Gastrocnemius/ soleus (ankle)	Plantar surface-foot Lateral aspect-foot	Gastrocnemius/ soleus (foot plantar flexion) Abductor hallucis (toe flexors) Gluteus maximus (hip extension)	Bottom foot, posterior calf, posterior thigh, buttocks	Straight leg–raising sign present

Brachial neuralgia /Brachial Neuritis

- Brachial neuritis (BN), also known as neuralgic amyotrophy or Parsonage-Turner syndrome, is a rare syndrome of unknown etiology affecting mainly the motor branches/fascicles of certain characteristic peripheral nerves in the arm.
- The term brachial may be a misnomer, as electrodiagnostic and radiologic evidence often points to a mononeuropathy or mononeuropathy multiplex rather than plexus involvement.
- Brachial neuritis usually is characterized by the acute onset of excruciating unilateral shoulder pain, followed by flaccid paralysis of shoulder and parascapular muscles several days later. The syndrome can vary greatly in presentation and nerve involvement.
- Physical therapy for patients with brachial neuritis should be focused on the maintenance of full range of motion (ROM) in the shoulder and other affected joints.

Symptoms of brachial neuritis

- The onset of pain in brachial neuritis (BN) is often abrupt and may follow recent illness, surgery, immunization, or even trauma. Up to two thirds of cases begin during the night time.
- The pain usually is localized to the right shoulder region, but it may be bilateral in 10-30% of cases. The pain's intensity is very high (9+/10) and is maximal at onset. Usually, the pain is described as sharp or throbbing in nature.

- The pain usually is constant, but it is exacerbated by movements of the shoulder. Movements of the neck, coughing, and/or sneezing usually do not worsen the pain.
- Intense pain can last from a few hours to several weeks and requires opiate analgesia.
- Low-grade pain may persist for up to a year. As pain subsides, weakness becomes apparent.
- Numbness may occur, depending on the particular nerves affected, and usually is found in the nerve distribution corresponding to maximal muscle weakness. However, numbness is rarely a prominent complaint.

Ancillary investigations

- Laboratory studies are indicated in brachial neuritis only if systemic disease is suspected on clinical grounds.
- The complete blood count (CBC) and erythrocyte sedimentation rate (ESR) can be used as nonspecific indicators of systemic disease.
- Antinuclear antibody (ANA) values can be used as a marker for connective-tissue disease.
- Human immunodeficiency virus (HIV) serology can also be performed.
- Magnetic resonance imaging (MRI) or computed tomography (CT) myelogram scanning should be considered initially to rule out cervical radiculopathy (particularly C5/C6).
- MRI of the brachial plexus can help to rule out carcinomatous or granulomatous infiltration, if clinically indicated.
- A shoulder radiograph may be indicated to rule out specific shoulder pathologies.
- Electrodiagnosis should be considered initially to confirm neuropathic diagnosis and to rule out various other conditions (eg, radiculopathy, neuropathy, amyotrophic lateral sclerosis).

Cervical spondylosis

Cervical spondylosis

1. Cervical spondylosis is the term given to the occurrence of osteoarthritis in the cervical spine.
2. It is characterised by degeneration of the intervertebral discs and osteophyte formation.
3. This extremely common and radiological changes of cervical spondylosis are very frequently found in apparently healthy individuals above the age of 50.
4. Whilst cervical spondylosis is often asymptomatic, it may be associated with neurological dysfunction.
5. The C5/6, C6/7 and C4/5 vertebral levels and C6, C7 and C5 roots, respectively, are most commonly affected.

Cervical spondylotic radiculopathy

Compression of a nerve root occurs when disc prolapses laterally, which may develop acutely, or compression may occur more gradually due to osteophytic encroachment of the intervertebral foramina.

Clinical features

1. The patient complains of pain in the neck that movement in the distribution of the affected nerve root.
2. The neck is held rigidly and neck movements may exacerbate pain.
3. Paraesthesia and sensory loss may be found in the affected segment
4. There may be lower motor neuron signs, including weakness, wasting and reflex impairment

Physical signs in cervical root compression

Root	Muscle weakness	Sensory loss	Reflex loss
C5	Biceps, deltoid, spinati	Upper lateral arm	Biceps
C6	Brachioradialis	Lower lateral arm, thumb, index finger	Supinator
C7	Triceps, finger and wrist extensors	Middle finger	Triceps

Investigations

1. Plain X-rays, including lateral and oblique views, should be obtained to confirm, the

presence of degenerative changes and to exclude other conditions, including destructive lesions.

2. If surgery is contemplated, MRI is required.
3. Electrophysiological studies rarely add to the clinical examination, but may be necessary if there is doubt about the differential diagnosis between root and peripheral nerve lesions.

Lumbar spondylosis (Spondylosis deformans, lumbar osteoarthritis)

- The term spondylolysis is derived from the Greek words spondylos, meaning vertebra, and lysis, meaning break or defect.
- It describes bony overgrowths (osteophytes, bone spurs), predominantly those at the anterior, lateral, and, less commonly, posterior aspects of the superior and inferior margins of vertebral centra(bodies).
- Lumbar spondylosis occurs as a result of new bone formation in areas where the anular ligament is stressed. The margins of vertebral bodies are normally smooth. Growth of new bone projecting horizontally at these margins identifies osteophytes.
- Most osteophytes are anterior or lateral in projection. Posterior vertebral osteophytes are less common and only rarely impinge upon the spinal cord or nerve roots. This dynamic process increases with, and is perhaps an inevitable concomitant, of age. Lumbar osteophytes have long been thought to cause back pain because of their frequency and size. This has led to many studies of the distribution of vertebral osteophytes, not all of which are pertinent. The frequency of signs or symptoms in individuals with osteophytes is no greater than that in individuals without osteophytes.
- Lumbosacral spondylolysis (lumbar spondylolysis) is a unilateral or bilateral defect of the pars interarticularis that affects one or more of the lumbar vertebrae
- *Spondylosis*, or osteoarthritic spine disease, typically occurs in later life and primarily involves the cervical and lumbosacral spine.
- Patients often complain of back pain that increases with movement and is associated with stiffness.
- The relationship between clinical symptoms and radiologic findings is usually not straightforward.
- Pain may be prominent when x-ray, CT, or MRI findings are minimal, and prominent degenerative spine disease can be seen in asymptomatic patients.
- Osteophytes or combined disk-osteophytes may cause or contribute to central spinal canal stenosis, lateral recess stenosis, or neural foraminal narrowing.

Epidemiology

Spondylolysis occurs in 6-10% of the general population and has been found to be as high as 25-60% in athletes. It is especially common in young athletes younger than 18 years who participate in sports that involve twisting or backward bending motions of the spine. This injury also runs in some families, suggesting that there may be a hereditary component

Clinical presentation

- Among the general population, most cases of lumbosacral spondylolysis (lumbar spondylolysis) are clinically inapparent, and the condition is symptomatic in only 10% of patients.
- Many athletes with lumbosacral spondylolysis (lumbar spondylolysis) are likewise asymptomatic.
- Lumbosacral spondylolysis (lumbar spondylolysis) typically occurs in young people, with a mean age at diagnosis in athletes of about 15-16 years.
- Patients with lumbosacral spondylolysis (lumbar spondylolysis) generally report low back pain aggravated by activity, particularly with hyperextension maneuvers such as in gymnastics.
- In general, patients present without a history of neurologic symptoms.

Physical Examination

The physical examination of the patient with lumbosacral spondylolysis (lumbar spondylolysis) frequently yields the following minimal findings:

- No tenderness to palpation is noted, but some discomfort can be elicited with deep percussion over the midline of the lumbar area.
- Range of motion is full. Because of the previously increased range of motion of dancers and gymnasts, they appear to have normal flexibility.
- Athletes suspected of having an injury must have their flexibility compared with that of their preinjury state.
- Forward flexion does not increase symptoms.
- Hyperextension mimicking the sporting movement generally elicits pain.
- Sciatica can occur but is rare.
- Physical findings that may also be present include antalgic or normal gait, tight lumbar musculature and hamstrings, hyperlordosis, and buttock or thigh pain.
- If a unilateral defect is present, the 1-leg hyperextension test elicits pain on the involved side. This test is performed by the patient bearing weight on one leg, with both the hip and knee of the other extremity flexed while hyperextending the lumbar spine.
- The maneuver is performed on both sides, and asymmetric low back pain indicates unilateral disease. Bilateral disease may show symmetric or asymmetric pain with this maneuver.
- The neurovascular examination findings are normal. The rest of the general low back pain examination must be performed, taking note of all dermatomes, myotomes, and reflexes.

Ancillary investigations

Imaging Studies

- Routine thoracolumbar radiography in an athlete suspected of having a lumbosacral spondylolysis (lumbar spondylolysis) should include anteroposterior (AP), lateral, and oblique views.
- Flexion and extension lateral radiographs can be added if spondylolisthesis is present or stability is in question.
- AP radiographic findings are usually normal in spondylolysis.
- The oblique views are particularly screened for the "Scotty dog" lesion in the pars interarticularis. The pars defect is represented by the collar on the Scotty dog. Keep in mind that the oblique view must be of good quality and read by someone experienced in making the diagnosis of lumbosacral spondylolysis (lumbar spondylolysis).
- An athlete who is symptomatic with hyperextension and whose plain radiography results are normal requires additional investigations. A 99-m technetium (99m Tc) ethylene diphosphonate bone scintigraphy identifies pars interarticularis stress fractures that were missed on oblique plain radiography. Those patients who have had recent trauma or strenuous activity and who are symptomatic show increased activity on the spondylolytic area. On the other hand, those with chronic low back pain show normal scans.
- Technetium bone scanning has its limitations. When compared to single-photon emission computed tomography (SPECT) scanning, the SPECT scanning was found to be more sensitive and better at localization of abnormalities in those athletes with radiographically obvious lumbosacral spondylolysis/spondylolisthesis.
- Bone scanning has a false-positive rate of 15% when using computed tomography (CT) scanning as the standard to establish the diagnosis of lumbosacral spondylolysis (lumbar spondylolysis). Therefore, SPECT scanning is needed to identify the more subtle stress reactions.

Sagittally reconstructed computed tomography scan

- Sagittally reconstructed computed tomography scan of the lumbar spine shows a defect of the pars interarticularis on the left at L5.
- CT scanning also provides information regarding other features of spondylolysis, such as facet joint changes, spondylolisthesis, and disc herniations, and it also helps prognosticate the potential for a lesion to heal.

- MRI is inferior to CT scanning for direct visualization of defects of the pars interarticularis. However, MRI has utility to rule out disc herniations.

Long TR (T2-weighted) fat suppressed sagittal magnetic resonance

- Long TR (T2-weighted) fat suppressed sagittal magnetic resonance image shows increased signal in the pars interarticularis on the left at L5
- This is an acute stress reaction.

Sagittal short TR (T1-weighted) magnetic resonance

Sagittal short TR (T1-weighted) magnetic resonance image shows decreased signal in the pars interarticularis on the left at L5

MCQs

1. Parkinson’s disease (PD) causes damage to the—— and is associated with impairments in habitlearning but spares ______
   a. Hippocampal episodic Memory
   b. Bilateral anterior temporal dysfunction Long-Term Memory
   c. Basal ganglia declarative memory
   d. Basal ganglia Nondeclarative Memory
2. Burning sensation in one half of the body (Hyperalgia) is seen in
   a. Thalamic stroke
   b. Lateral medullary syndrome
   c. Cerebellar lesion
   d. Meningitis
3. Which cranial nerve is affected in Bell’s palsy?
   a. Trigeminal nerve (Cranial nerve V)
   b. Facial nerve (Cranial nerve VII)
   c. Vagus nerve (Cranial nerve X)
   d. All of the above
4. The upper motor neuron impairment produces the following change of muscles tone:
   a. Flaccidity
   b. Spasticity
   c. “Cog wheel” rigidity
   d. Myoclonia
5. The muscular wasting (hypotrophy) usually develops with disease in:
   a. Upper motor neuron
   b. Lower motor neuron
   c. Cerebellar
   d. Caudate
6. The ability to walk along a straight line is most often impaired with:
   a. Cerebellar dysfunction
   b. Parietal lobe damage
   c. Temporal lobe damage
   d. Ocular motor disturbances
7. Parkinsonism includes combination of the following:
   a. Tremor, bradykinesia& muscles rigidity
   b. Paresis, anesthesia & muscles spasticity
   c. Chorea & muscles hypotonia
   d. Tremor, ataxia & muscles hypotonia
8. Meningeal sign is the following:
   a. Babinsky
   b. Kernig
   c. Lasseg
   d. Romberg
9. ElevatedIgG index level in CSF is seen in which among the following disease
   a. Acute transverse myelitis
   b. Syringomyelia
   c. Conusmedullaris syndrome
   d. Spinal cord abscess
10. Parsonage-Turner syndrome is also known as
    a. Brachial neuritis
    b. Amyotrophic Lateral Sclerosis
    c. Multiple sclerosis
    d. Parkinson’s disease

Answer

1.	c	2.	a	3.	b
4.	b	5.	b	6.	a
7.	a	8.	b	9.	a
10.	a				

CHAPTER

43 Sandhigata Vata and Asthi Majjagata Vata

सन्धिगत वात (Sandhigata Vāta)

Vata aggravating etiological factors

रुक्षशीताल्पलघ्वन्नव्यवायातिप्रजागरैः ।
विषमादुपचाराच्च दोषासृक्स्त्रवणादति ॥
लङ्घनप्लवनात्यध्वव्यायामातिविचेष्टितैः ।
धातूनां संक्षयाच्चिन्ताशोकरोगातिकर्षणात् ॥
दुःखशय्यासनात् क्रोधाद्दिवास्वप्नाद्भयादपि ।
वेगसंधारणादामादभिघातादभोजनात् ॥
मर्माघाताद्गजोष्ट्राश्वशीघ्रयानापतंसनात् ।
C.S.Ci. 28/15-18

Vāta gets aggravated by adopting following *Nidānas* such as- excessive consumption of ununctous, cold, scanty, & light food; excessive sexual intercourse; excess vigilance; improper therapeutic procedures; administration of therapies which causes excessive elimination of *Doṣas* and *Rakta*, excess fasting; excessive movements such as leaping, jumping, warfaring and physical exercise; depletion of *Dhātus*; excessive emaciation due to worry, grief and affliction of disease; sleeping over uneven beds & sitting in odd postures; anger; day sleep, fear and suppression of natural urges; *Āma doṣa*; trauma; abstinence from food; injuries to vital body parts; falling down from elephants, horses and other fast moving vehicles.

तत्र बलवद्विग्रहातिव्यायामव्यवायाध्ययनप्रपतनप्रधावनप्रपीडनाभिधातलङ्घनप्लवनतरणरात्रिजागरणभारहरणगजतुरङ्गरथपदाति चर्य्याकटुकषायतिक्तरूक्षलघुशीतवीर्य्यशुष्कशाकवल्लूरवरकोद्दालककोरदूषश्यामाकनीवारमुद्गमसूराढकीहरेणुकलायनिष्पावानशनविषमाशनाध्यशनवातमूत्रपुरीषशुक्रच्छर्दिक्षवथूद्गारवाष्पवेगविघातादिभिर्विशेषैर्वायुः प्रकोपमापद्यते ॥ *S.S.Su.21/19*

Suśruta has described the etiological factors which are responsible for vitiation of *Vāta doṣa* such as fighting with stronger person, excessive exercise, sexual intercourse, excessive study, falling down, excessive running, excessive pressurizing by feet, trauma, light diet, excessive swimming, excessive hopping, night awakening, suppression natural urges etc.

Vāta gets aggravated by two different causes viz-

1. *Mārgāvaraṇajanya*: by the occlusion of channels, *it is also called as upstambhit* and
2. *Dhātukṣayajanya* : by the depletion of *dhātus*, it is also called as *nir-upastambhit*

वायोर्धातुक्षयात् कोपो मार्गस्यावरणेन च (वा)।
C.S.Ci. 28/59

Treatment of these two types are nearly opposite in nature. Therefore, understanding of these two types are important at the time of management.

Clinical presentation

वातपूर्णदृतिस्पर्शः शोथः सन्धिगतेऽनिले ।
प्रसारणाकुञ्चनयोः प्रवृत्तिश्च सवेदना ॥
C.S.Ci. 28/37

Excessively aggravated *Vāta* inside joints causes swelling in the joints, appears as a leather bag inflated with air on palpation and pain during extension & flexion of the joints.

हन्ति सन्धिगतः सन्धीञ्शूलशोफौ करोति च।
S.S.Ni 1/28

Excessively aggravated *Vāta* inside joints causes the destruction of functions of joints and gives rise to pain & swelling in the joints.

हन्ति सन्धिगतः सन्धीन् शूलाटापौ करोति च ।
M.Ni. 22/21

Excessively aggravated *Vāta* in the joints causes destruction of the actions of joints and manifest pain & crepitation in the joints.

Pathological components

Doṣa - *Vāta pradhāna*
Dūṣya - *Rasa, Asthi, Snāyu, Kaṇḍrā, Sirā*

Adhiṣṭhāna - *Sandhipradeśa*

Srotas - *Rasa, Asthi and Majjāvaha srotas*

Types of Srotoduṣṭi - *Saṅga*

Nature of disease - *Chronic*

Treatment - Difficult to be cure

Classification of *Sandhigata Vāta*

ये वातव्याधयः सन्धीनाश्रयन्ति शनैः शनैः।
सद्यो वा शूलशोफाभ्यां सन्धिवात हि ते मताः॥
रसवातो रक्तवातो विषवातस्तथापरः।
जीर्णवातो जरावातः पञ्च स्युस्तद्विदा इहा॥
Si.Ni. 8/513-514

Due to consumption of etiological factors *Vāta* gets infuriated and progressively trouble the *Sandhi* (joints) and produces the *Sandhi Śūla* (pain in the joints) and *Sandhi śopha* (swelling of the joints).

Sandhivāta is classified into five types namely *Rasavāta, Raktavāta, Viṣavāta. Jirṇavāta* and *Jarāvāta*.

Rasavāta: *Rasavāta* is characterised by *Ruja* (pain) and *Śopha* (swelling) in *Sākhā, Trika, Kaṭi, Pṛṣṭha, Grīva sandhīs, Sarvagātrapīḍa* (pain all over the body) accompanied with *Avipāka* (indigestion), *Aruci* (anorexia/loss of appetite) and *Alpajvara vijvaroathavā* (mild fever or no fever). It is also known as *Āmavāta*.

Raktavāta: *Raktavāta* is characterised by *Rugdāharāgayukta śotha* (swelling associated with pain, burning sensation and redness), *Sandhau ya śīghrasañcara* (migratory pain moves quickly from one joint to another) and *Caraṇāṅguṣṭhamūlādau* (especially affects the root of the big toe)

***Viṣavāta*:** *Viṣavāta* is characterised by *Ruja* (pain) and *Śopha* (swelling) in *Jānvaṃsakaṭipṛṣṭhādau* (knee, shoulder, waist, back etc.,) due to *Phiraṅga* and *Pūyamehādi viṣa*

***Jīrṇavāta*:** Progressive Sandhivāta affects *Nānāsandhisamāśraya* (many joints) and *Niścalīkurute sandhīn* (makes them immovable), it is known as *Jīrṇavāta* & it is chronic condition.

Jarāvāta: *Jarāvāta* is an *Asthi vikṛti* (bone disorder) mainly affecting the *Pṛṣṭhavaṃśā* (back and shoulder) and causes *Kubjatā* (state of being humpbacked) due to *Jarāvastha* (old age).

कटी ग्रह (*Kaṭi Graha*)

वायुः कट्याश्रितः शुद्धः सामो वा जनयेद्रुजम्।
कटिग्रहः स विज्ञेयः पंगुःसक्थि द्वयश्रितः।
Gadanigraha"

- The exacerbated vata reaches the lower back region and takes shelter there and produces *Kaṭigraha.*
- *Vāta doṣa* either alone or in association with *āma* produce symptoms and signs
- It is characterised by pain and restricted movement in the low back region.
- Patients find it difficult to move limbs, walk, and bend.

Exposure to etiological factors
↓
Exacerbation of *vāta doṣa*
↓
Reaches low back region
↓
Localisation of morbid factors causing *doṣa-dūṣya* amalgamation
↓
In certain cases *doṣa* amalgamate with *āma*
↓
Kaṭi Graha

सम्प्राप्ति घटक (*Samprāpthi gaṭaka)*

Doṣa	*Vāta, Vāta Kapha*
Dūṣya	*Asthi, Kaṇḍara, Snāyu*
Udbhava Sthāna	*Pakvāśaya*
Srotas	*Māṃsavaha, Asthivaha*
	Srotoduṣṭi Sanga
Agni	*Jaṭarāgni, Dhātvagni*
Ama	*Jaṭarāgni & Dhātvagni-mandya Janya Āma*
Rogamārga	*Madhyamā*
Vyaktasthāna	*Kaṭi*

मन्यास्तम्भ-Manyāstambha

Etio-pathogenesis: Sleeping during day time, improper posture and improper way of sitting, standing & gazing upwards leads to aggravation of *Vāta* and enveloped by *Kapha* produces *Manyāstambha.*

दिवास्वन्पासनस्थानविवृतोर्ध्वनिरीक्षणैः ।
मन्यास्तम्भं प्रकुरुते स एव श्लेष्मणाऽऽवृतः ॥
S.S.Ni 1/67

Clinical features

Following features are commonly pointed out by the patient.

1. Stretching in the sternocleidomastoid muscles.
2. Stiffness of above muscles and rest of the other muscles
3. Severe pain in the neck region.

वातकण्टक (Vāta Kaṇṭaka)

रुक् पादे विषमन्यस्ते श्रमाद्वा जायते यदा।
वातेन गुल्फमाश्रित्य तमाहुर्वातकण्टकम् ॥
A.H.Ni. 15/53

न्यस्ते तु विषमं पादे रुजः कुर्य्यात् समीरणः ।
वातकण्टक इत्येष विज्ञेयः खुडुकाश्रितः ॥
S.S.Ni 1/79

While walking when the foot is placed unevenly on the ground, *Vāta doṣa* is vitiated and develops sprains at the ankle joint/joints of feet, which is associated with severe pain and swelling. This is also known as *khuḍḍavāta* or *pādakaṇṭaka*

अवबाहु एवं अंशशोष (Avabāhuka and Ansaśoṣa)

Clinical features

Aggravated *Vāta* in shoulder region dries up its binding elements and constrict the *Sirās* of the shoulder region is known as *Avabāhuka.*

असंदेशस्थितो वायुः शोषयित्वांऽसबन्धनम् ।
शिराश्चाकुञ्च्य तत्रस्थो जनयत्यवबाहुकम् ॥
S.S.Ni 1/82

Aggravated *vāta*, dries up the binding ligament, tendons and muscles in the shoulder region may lead to develop *Aṅsaśoṣa*.

CHAPTER

44

Diseases of Bone, Joints and Muscles

Osteoarthritis

Osteoarthritis (OA) is the most common non-inflammatory arthritis. Its high prevalence is high in the elderly, and the high rate of disability related to disease makes it as a leading cause of disability. The occurrence of osteoarthritis is on the rise due to faulty lifestyle. Obesity specially imparts significant contribution to the weight bearing joints. Commonly affected joints include the cervical and lumbosacral spine, hip, knee, and first metatarsal phalangeal joint (MTP). In the hands, the distal and proximal interphalangeal joints and the base of the thumb are often affected. Usually affected joints are the wrist, elbow, and ankle. The symptoms can interfere with work and normal daily activities. Unlike some other types of arthritis, if not affects the internal organs. Among those over 60 years old, about 10% of males and 18% of females are affected with OA. In the affected individual, osteoarthritis is the cause of about 2% ofyears lived with disability.

Osteoarthritis at a glance

The formation of hard knobs at the middle finger joints (known as Bouchard's nodes) and at the farthest joints of the fingers (known as Heberden's nodes) are a common feature of osteoarthritis in the hands.

Age group : Commonly observed in elderly age group

Symptoms : Joint pain, stiffness, joint swelling, joint creptus and decreased range of motion

Usual onset : Over years

Causes : Previous joint injury, abnormal joint or limb development, inherited factors.

Risk factors : Overweight, legs of different lengths, job with high levels of joint stress.

Diagnostic method : Based on symptoms, supported by other testing.

Treatment : Exercise, efforts to decrease joint stress, support groups, pain medications, joint replacement.

Frequency : 237 million / 3.3% (2015)

Definition

Osteoarthritis is the most common type of joint disease, affecting many people in the world. It can be thought of as a degenerative disorder arising from the biochemical breakdown of articular (hyaline) cartilage in the synovial joints. However, the current view holds that osteoarthritis involves not only the articular cartilage but also the entire joint organ, including the subchondral bone and synovium. OA is joint failure, a disease in which all structures of the joint have undergone pathologic change, often in concert. The pathologic sine qua non of disease is hyaline articular cartilage loss, present in a focal and, initially, nonuniform manner. This is accompanied by increasing thickness and sclerosis of the subchondral bony plate, by outgrowth of osteophytes at the joint margin, by stretching of the articular capsule, by mild synovitis in many affected joints, and by weakness of muscles bridging the joint. In knees, meniscal degeneration is part of the disease. There are numerous pathways that lead to joint failure, but the initial step is often joint injury in the setting of a failure of protective mechanisms.

Aetiology of OA

The daily stresses applied to the joints, especially the weight-bearing joints (eg, ankle, knee, and hip), play an important role in the development of osteoarthritis. External forces accelerate the catabolic effects of the chondrocytes and further disrupt the cartilaginous matrix.

Risk factors for osteoarthritis include the following:

- Age

- Obesity
- Trauma
- Genetics (significant family history)
- Reduced levels of sex hormones
- Muscle weakness
- Repetitive use (ie, jobs requiring heavy labor and bending)
- Infection
- Crystal deposition
- Acromegaly
- Previous inflammatory arthritis (eg, burnt-out rheumatoid arthritis)
- Heritable metabolic causes (eg, alkaptonuria, hemochro-matosis, and Wilson disease)
- Hemoglobinopathies (eg, sickle cell disease and thalassemia)
- Neuropathic disorders leading to a Charcot joint (eg, syringomyelia, tabes dorsalis, and diabetes)
- Underlying morphologic risk factors (eg, congenital hip dislocation and slipped femoral capital epiphysis)
- Disorders of bone (eg, Paget disease and avascular necrosis)
- Previous surgical procedures (eg, meniscectomy)

Advancing age

With advancing age come reductions in cartilage volume, proteoglycan content, cartilage vascularization, and cartilage perfusion.

Obesity

Obesity increases the mechanical stress in a weight-bearing joint. It has been strongly linked to osteoarthritis of the knees and, to a lesser extent, of the hips.Obesity is associated with increased levels (both systemic and intra-articular) of adipokines (cytokines derived from adipose tissue), which may promote chronic, low-grade inflammation in joints.

Other causes

Trauma or surgery (including surgical repair of traumatic injury) involving the articular cartilage, ligaments, or menisci can lead to abnormal biomechanics in the joints and accelerate osteoarthritis.

Muscle dysfunction compromises the body's neuromuscular protective mechanisms, leading to increased joint motion and ultimately resulting in osteoarthritis. Valgus malalignment at the knee has been shown to increase the incidence and risk of radiographic progression of knee osteoarthritis involving the lateral compartment.

Genetics

A hereditary component, particularly in osteoarthritis presentations involving multiple joints, has long been recognized.

Patho-physiology

The pathology of OA provides evidence of the involvement of many joint structures in disease. Cartilage initially shows surface fibrillation and irregularity. As disease progresses, focal erosions develop there, and these eventually extend down to the subjacent bone. With further progression, cartilage erosion down to bone expands to involve a larger proportion of the joint surface, even though OA remains a focal disease with non-uniform loss of cartilage.

After an injury to cartilage, chondrocytes undergo mitosis and clustering. While the metabolic activity of these chondrocyte clusters is high, the net effect of this activity is to promote proteoglycan depletion in the matrix surrounding the chondrocytes. This is because the catabolic is greater than the synthetic activity. As disease develops, collagen matrix becomes damaged, the negative charges of proteoglycans get exposed, and cartilage swells from ionic attraction to water molecules. Because in damaged cartilage proteoglycans are no longer forced into close proximity, cartilage does not bounce back after loading as it did when healthy, and cartilage becomes vulnerable to further injury. Chondrocytes at the basal level of cartilage undergo apoptosis.

With loss of cartilage come alterations in subchondral bone. Stimulated by growth factors and cytokines, osteoclasts and osteoblasts in the subchondral bony plate, just underneath cartilage, become activated. Bone formation produces a thickening and stiffness of the subchondral plate that occurs even before cartilage ulcerates. Trauma to bone during joint loading may be the primary factor driving this bone response, with healing from injury (including microcracks) producing stiffness. Small areas of osteonecrosis usually exist in joints with advanced disease. Bone death may also be caused by bone trauma with shearing of microvasculature, leading to a cutoff of vascular supply to some bone areas.

At the margin of the joint, near areas of cartilage loss, osteophytes form. These starts as outgrowths of new cartilage and, with neurovascular invasion from the bone, this cartilage ossifies. Osteophytes are an important radiographic hallmark of OA. In malaligned joints, osteophytes grow larger on the side of the joint subject to most loading stress (e.g., in varus knees, osteophytes grow larger on the medial side).

The synovium produces lubricating fluids that minimize shear stress during motion. In healthy joints, the synovium consists of a single discontinuous layer filled with fat and containing two types of cells, macrophages and fibroblasts, but in OA, it can sometimes become oedematous and inflamed. There is a migration of macrophages from the periphery into the tissue, and cells lining the synovium proliferate. Enzymes secreted by the synovium digest cartilage matrix that has been sheared from the surface of the cartilage.

Additional pathologic changes occur in the capsule, which stretches, becomes oedematous, and can become fibrotic.

Classification of Arthritis

A number of classification systems are used for gradation of osteoarthritis, some important are given as below.

- WOMAC scale, taking into account pain, stiffness and functional limitation.
- Kellgren-Lawrence grading scale for osteoarthritis of the knee. It uses only projectional radiographyfeatures.
- Tinnis classification for osteoarthritis of the hip joint, also using only projectional radiography features.
- Knee injury and Osteoarthritis Outcome Score (KOOS) and Hip disability and Osteoarthritis Outcome Score (HOOS) surveys.

Osteoarthritis can be classified into either primary or secondary depending on whether or not there is an identifiable underlying cause. Both primary generalized nodal osteoarthritis and erosive osteoarthritis (EOA, also called inflammatory osteoarthritis) are sub-sets of primary osteoarthritis. EOA is a much less common, and more aggressive inflammatory form of osteoarthritis which often affects the distal inter-phalangeal joints of the hand and has characteristic articular erosive changes on x-ray

Osteoarthritis can be classified on the basis of joint involvement:

- Hand:
  o Trapeziometacarpal osteoarthritis
- Wrist (wrist osteoarthritis)
- Vertebral column(spondylosis)
  o Facet joint arthrosis
- Hip osteoarthritis
- Knee osteoarthritis

Clinical Features

- Joint pain from OA is activity-related. Pain comes on either during or just after joint use and then gradually resolves. Early in disease, pain is episodic, triggered often by a day or two of overactive use of a diseased joint, such as a person with knee OA taking a long run and noticing a few days of pain thereafter. As disease progresses, the pain becomes continuous and even begins to be bothersome at night. Stiffness of the affected joint may be prominent, but morning stiffness is usually brief (<30 min).
- In knees, buckling may occur, in part, due to weakness of muscles crossing the joint. Mechanical symptoms, such as buckling, catching, or locking, could also signify internal derangement, such as meniscal tears, and need

to be evaluated. In the knee, pain with activities requiring knee flexion, such as stair climbing and arising from a chair, often emanates from the patellofemoral compartment of the knee, which does not actively articulate until the knee is bent 35ã.Bursitis occurs commonly around knees and hips.

Physical Examination

Physical examination findings in patients with osteoarthritis are mostly limited to the affected joints. Following are the most common physical finding noted in cases of OA.

- Tenderness is over the affected joint line
- Reduced range of motion
- Crepitus are frequently present.
- Malalignment with a bony enlargement may occur.
- Bland effusion may be present over involved joints.
- Limitation of joint motion or muscle atrophy around a more severely affected joint may occur.
- Heberden nodes, which represent palpable osteophytes in the DIP joints, are more characteristic in women than in men.
- Inflammatory changes are mostly absent or at least not pronounced.

Progression of Osteoarthritis

The etiopathogenesis of osteoarthritis has been divided into 3 stages as follows:

- Stage 1 – Proteolytic breakdown of the cartilage matrix occurs
- Stage 2 – Fibrillation and erosion of the cartilage surface develop, with subsequent release of proteoglycan and collagen fragments into the synovial fluid
- Stage 3 – Breakdown products of cartilage induce a chronic inflammatory response in the synovium, which in turn contributes to further cartilage breakdown.

Ancillary Investigations

- Osteoarthritis is typically diagnosed on the basis of clinical and radiographic evidence. No specific laboratory abnormalities are associated with osteoarthritis.
- Levels of acute-phase reactants are typically within the reference range in patients with osteoarthritis. The erythrocyte sedimentation rate (ESR) is not usually elevated, though it may be slightly so in cases of erosive inflammatory arthritis. The synovial fluid analysis usually shows a white blood cell (WBC) count below 2000/L, with a mononuclear predominance.

Osteoporosis

Osteoporosis is a silent disease that causes no symptoms until a fracture occurs. It is a major public health concern, with about 44 million American men and women, or 55% of the population age 50 and over, having osteoporosis or low bone density that can lead to fractures. About 80% of osteoporosis occurs in women and 20% in men. The prevalence is increasing, with over 61 million expected to have osteoporosis or low bone density by 2020. About 30% of Caucasian women age 50 and over have osteoporosis, when defined as a T-score of less than -2.5 at the spine, hip, or mid-forearm. In men age 50 and older, the prevalence of osteoporosis is about 19%. The Dubbo study found that at the age 60 there was a residual lifetime fracture risk of 56% for women and 29% for men, assuming average life expectancy. A woman's risk of hip fracture is equal to her combined risk of breast, uterine, and ovarian cancer. Fractures of the spine and hip are associated with an increased risk of chronic pain, deformity, depression, disability, and death. About 50% of those with a hip fracture will be permanently unable to walk without assistance and 25% will require long-term care. The mortality rate five years after a fracture of the hip or a clinical vertebral fracture is about 20% greater than expected, with men having higher mortality rates than women.

Risk factors

Risk factors for osteoporotic fracture can be split between non-modifiable and modifiable. In addition, osteoporosis is a recognized complication of specific diseases and disorders. Medication use is theoretically modifiable, although in many cases, the use of medication that increases osteoporosis risk may be unavoidable. Caffeine is not a risk factor for osteoporosis. It is more common in females than males.

Non-modifiable risk factors

Bone density peaks at about 30 years of age. Women lose bone mass more rapidly than men.

- The most important risk factors for osteoporosis are advanced age (in both men and women) and female sex; oestrogen deficiency following menopause or surgical removal of the ovaries is correlated with a rapid reduction in bone mineral density, while in men, a decrease in testosterone levels has a comparable (but less pronounced) effect.
- **Ethnicity**: While osteoporosis occurs in people from all ethnic groups, European or Asian ancestry predisposes for osteoporosis.
- **Heredity**: Those with a family history of fracture or osteoporosis are at an increased risk; the heritability of the fracture, as well as low bone mineral density, is relatively high, ranging from 25 to 80%. At least 30 genes are associated with the development of osteoporosis.
- Those who have already had a fracture are at least twice as likely to have another fracture compared to someone of the same age and sex.
- **Build**: A small stature is also a non-modifiable risk factor associated with the development of osteoporosis.

Modifiable Factors

- Excessive alcohol: Although small amounts of alcohol are probably beneficial (bone density increases with increasing alcohol intake), chronic heavy drinking of probably increases fracture risk despite any beneficial effects on bone density.
- **Vitamin D deficiency:** Low circulating Vitamin D is common among the elderly worldwide. Mild vitamin D insufficiency is associated with increased parathyroid hormone (PTH) production. PTH increases bone resorption, leading to bone loss. A positive association exists between serum 1,25-dihydroxycholecalciferol levels and bone mineral density, while PTH is negatively associated with bone mineral density.
- **Tobacco smoking:** Tobacco smoking has been assumed to inhibit the activity of osteoblasts, and is an independent risk factor for osteoporosis. Smoking also results in increased breakdown of exogenous estrogen, lower body weight and earlier menopause, all of which contribute to lower bone mineral density.
- **Malnutrition:** Nutrition has an important and complex role in maintenance of good bone. Identified risk factors include low dietary calcium and/or phosphorus, magnesium, zinc, boron, iron, fluoride, copper, vitamins A, K, E and C (and D where skin exposure to sunlight provides an inadequate supply). Excess sodium is a risk factor. High blood acidity may be diet-related, and is a known antagonist of bone. Some have identified low protein intake as associated with lower peak bone mass during adolescence and lower bone mineral density in elderly populations. Conversely, some have identified low protein intake as a positive factor, protein is among the causes of dietary acidity. Imbalance of omega-6 to omega-3 polyunsaturated fats is yet another identified risk factor.
- **High dietary protein from animal sources**: A diet high in plant protein may be optimal for bone health, as higher protein diets tend to increase absorption of calcium from the diet and are associated with higher bone density. Indeed, it has recently been argued that low protein diets cause poor bone health.
- **Underweight/inactive:** Bone remodelling occurs in response to physical stress, so physical inactivity can lead to significant bone loss. Weight bearing exercise can increase peak bone mass achieved in adolescence, and a highly significant correlation between bone strength and muscle strength has been

determined. The incidence of osteoporosis is lower in overweight people.

- **Endurance training:** In female endurance athletes, large volumes of training can lead to decreased bone density and an increased risk of osteoporosis. However, for male athletes, the situation is less clear, and although some studies have reported low bone density in elite male endurance athletes, others have instead seen increased leg bone density.
- **Heavy metals:** A strong association between cadmium and lead with bone disease has been established. Low-level exposure to cadmium is associated with an increased loss of bone mineral density readily in both genders, leading to pain and increased risk of fractures, especially in the elderly and in females. Higher cadmium exposure results in osteomalacia (softening of the bone).
- **Soft drinks:** Recent studies reveal that soft drinks may increase risk of osteoporosis, at least in women· Others suggest soft drinks may displace calcium-containing drinks from the diet rather than directly causing osteoporosis.
- Proton pump inhibitors (such as lansoprazole, esomeprazole, or omeprazole) that decrease stomach acid, are a risk for bone fractures if taken for two or more years, due to decreased absorption of calcium in the stomach.

Medical disorders may lead to Osteoporosis

The body regulates calcium homeostasis with two pathways; one is signalled to turn on when blood calcium levels drop below normal and one is the pathway that is signalled to turn on when blood calcium levels are elevated.

Many diseases and disorders have been associated with osteoporosis. Some important one is given below.

- In general, immobilization causes bone loss.
- Hypogonadal states can cause secondary osteoporosis.
- Endocrine disorders that can induce bone loss include Cushing's syndrome, hyperparathyroidism, hyperthyroidism, hypothyroidism, diabetes mellitus type 1 and 2, acromegaly, and adrenal insufficiency.
- Malnutrition, parenteral nutrition and malabsorption can lead to osteoporosis. People with lactose intolerance or milk allergy may develop osteoporosis due to restrictions of calcium-containing foods. Individuals with bulimia can also develop osteoporosis. Those with an otherwise adequate calcium intake can develop osteoporosis due to the inability to absorb calcium and/or vitamin D. Other micronutrients such as vitamin K or vitamin B_{12} deficiency may also contribute.
- People with rheumatologic disorders such as rheumatoid arthritis, ankylosing spondylitis, systemic lupus erythematosus and polyarticular juvenile idiopathic arthritis are at increased risk of osteoporosis.
- Chronic kidney disease can lead to renal osteodystrophy.
- Hematologic disorders linked to osteoporosis are multiple myelomaand other monoclonal gammopathies, lymphoma, leukemia, mastocytosis, hemophilia, sickle-cell disease and thalassemia.
- Several inherited disorders have been linked to osteoporosis. These include osteogenesis imperfecta, Marfan syndrome, hemochromatosis, etc.
- People with scoliosis of unknown cause also have a higher risk of osteoporosis. Bone loss can be a feature of complex regional pain syndrome. It is also more frequent in people with Parkinson's disease and chronic obstructive pulmonary disease.
- People with Parkinson's disease have a higher risk of broken bones. This is related to poor balance and poor bone density. In Parkinson's disease there may be a link between the loss of dopaminergic neurons and altered calcium metabolism.

Medication may lead to Osteoporosis

Certain medications have been associated with an increase in osteoporosis risk, which are described as below.

- Steroid-induced osteoporosis (SIOP) arises due to use of glucocorticoids – analogous to Cushing's syndrome and involving mainly the axial skeleton. The synthetic glucocorticoid prescription drug prednisone is a main candidate after prolonged intake.

- Barbiturates, phenytoin and some other enzyme-inducing antiepileptics – these probably accelerate the metabolism of vitamin D.
- L-Thyroxine over-replacement may contribute to osteoporosis, in a similar fashion as thyrotoxicosis does. This can be relevant in subclinical hypothyroidism.
- Several drugs induce hypogonadism, for example aromatase inhibitors used in breast cancer, methotrexate and other antimetabolite drugs, depot progesterone and gonadotropin-releasing hormone agonists.
- Anticoagulants – long-term use of heparin is associated with a decrease in bone density, and warfarin (and related coumarins) have been linked with an increased risk in osteoporotic fracture in long-term use.
- Proton pump inhibitors – these drugs inhibit the production of stomach acid; this is thought to interfere with calcium absorption. Chronic phosphate binding may also occur with aluminium-containing antacids.
- Thiazolidinediones (used for diabetes) – rosiglitazone and possibly pioglitazone, inhibitors of PPARλ, have been linked with an increased risk of osteoporosis and fracture.
- Chronic lithium therapy has been associated with osteoporosis.

Signs and symptoms

Illustration depicting normal standing posture and osteoporosis

Osteoporosis itself has no symptoms; its main consequence is the increased risk of bone fractures. Osteoporotic fractures occur in situations where healthy people would not normally break a bone; they are therefore regarded as fragility fractures. Typical fragility fractures occur in the vertebral column, rib, hip and wrist.

1. Fractures

Fractures are a common symptom of osteoporosis and can result in disability. Acute and chronic pain in the elderly is often attributed to fractures from osteoporosis and can lead to further disability and early mortality. These fractures may also be asymptomatic. The most common osteoporotic fractures are of the wrist, spine, shoulder and hip. The symptoms of a vertebral collapse are sudden back pain, often with radicular pain (shooting pain due to nerve root compression) and rarely with spinal cord compression or cauda equina syndrome. Multiple vertebral fractures lead to a stooped posture, loss of height, and chronic pain with resultant reduction in mobility.

Fractures of the long bones acutely impair mobility and may require surgery. Hip fracture, in particular, usually requires prompt surgery, as serious risks are associated with it, such as deep vein thrombosis and pulmonary embolism, and increased mortality.

Fracture risk calculators assess the risk of fracture based upon several criteria, including bone mineral density, age, smoking, alcohol usage, weight, and gender. The term "established osteoporosis" is used when a broken bone due to osteoporosis has occurred. Osteoporosis is a part of frailty syndrome.

Risk of fall

There is an increased risk of falls associated with aging. These falls can lead to skeletal damage at the wrist, spine, hip, knee, foot, and ankle. Part of the fall risk is because of impaired eyesight due to glaucoma, & macular degeneration, balance disorder, movement disorders, dementia, and sarcopenia (age-related loss of skeletal muscle). Collapse (transient loss of postural tone with or without loss of consciousness).

Pathogenesis

The underlying mechanism in all cases of osteoporosis is an imbalance between bone resorption and bone formation. In normal bone, matrix remodeling of bone is constant; up to 10% of all bone mass may be undergoing remodeling at any point in time. The process takes place in bone multicellular units (BMUs) as first described by Frost & Thomas in 1963. Osteoclasts are assisted by transcription factor PU. to degrade the bone matrix, while osteoblasts rebuild the bone matrix. Low bone mass density can then occur when osteoclasts are degrading the bone matrix faster than the osteoblasts are rebuilding the bone.

The three main mechanisms by which osteoporosis develop are an inadequate peak bone mass (the skeleton develops insufficient mass and strength during growth), excessive bone resorption, and inadequate formation of new bone during remodeling. Interplay of these three mechanisms underlies the development of fragile bone tissue.

Hormonal factors strongly determine the rate of bone resorption; lack of estrogen (as a result of menopause) increases bone resorption, as well as decreasing the deposition of new bone that normally takes place in weight-bearing bones. The amount of estrogen needed to suppress this process is lower than that normally needed to stimulate the uterus and breast gland. The í-form of the estrogen receptor appears to be the most important in regulating bone turnover.

In addition to estrogen, calcium metabolism plays a significant role in bone turnover, and deficiency of calcium and vitamin D leads to impaired bone deposition; in addition, the parathyroid glands react to low calcium levels by secreting parathyroid hormone (parathormone, PTH), which increases bone resorption to ensure sufficient calcium in the blood. The role of calcitonin, a hormone generated by the thyroid that increases bone deposition, is less clear and probably not as significant as that of PTH.

Adhesive Capsulitis (Frozen Shoulder)

- Adhesive capsulitis and frozen shoulder syndrome (FSS) are two terms that have been used to describe a painful and stiff shoulder.
- The current consensus definition of a frozen shoulder by the American Shoulder and Elbow Surgeons is "a condition of uncertain etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder."
- The American Academy of Orthopaedic Surgeons defines this condition as: "A condition of varying severity characterized by the gradual development of global limitation of active and passive shoulder motion where radiographic findings other than osteopenia are absent."
- The loss of passive range of motion (ROM) is a critical element in establishing the diagnosis of a true frozen shoulder. Although conditions such as subacromial bursitis, calcifying tendinitis, and partial rotator cuff tears can be associated with significant pain and loss of active ROM, passive ROM is preserved. Therefore, patients with those conditions should not be classified as having a frozen shoulder.
- Patients with primary frozen shoulder have no significant findings in the history, clinical examination, or radiographic evaluation to explain their motion loss and pain. Classically, symptoms of primary frozen shoulder have been divided into three phases: freezing (painful), frozen (stiffening), and thawing.

Stages of Frozen Shoulder Syndrome

Stage	Duration
Stage 1 Freezing (painful)	Up to 9 months
Stage 2 Frozen (stiffening)	4 months - 20 months
Stage 3 Thawing	5 months- 26 months
Contralateral shoulder	6 months-7 years after initial onset of symptoms

- Patients with diabetes and younger patients may be more likely to develop contralateral idiopathic adhesive capsulitis.

In contrast to patients with primary FSS, patients with secondary FSS describe an event that preceded the onset of shoulder symptoms, such as the following:

- Upper extremity trauma (eg, shoulder surgery, rotator cuff tear, proximal humerus facture)
- Immobilization (eg, cardiothoracic surgery, neurosurgery)
- Metabolic/endocrine (eg, thyroid disease, diabetes mellitus, autoimmune disease, hyperlipidemia)
- Neurologic (eg, stroke, Parkinson disease)
- Cardiac disease (eg, ischemic heart disease, hypertension)
- Drugs (eg, protease inhibitors, antiretrovirals, immunizations, fluoroquinolones)
- Malignancy

Etiology

- Duplay was one of the first physicians to present the concept of periarticular tissue pathology rather than periarticular arthritis as the cause of frozen shoulder.
- Despite a lack of evidence linking frozen shoulder to a specific etiology, various triggers that may predispose patients to this problem appear to exist

A few reported etiologic agents include the following:

- Trauma
- Surgery (including but not limited to shoulder surgery)
- Inflammatory disease
- Diabetes
- Regional conditions
- Various shoulder maladies

Finally, frozen shoulder also has been associated with the following disorders:

- Cervical spine disease
- Parkinson disease
- Hyperthyroidism
- Hypothyroidism
- Ischemic heart disease

Clinical Presentation

Patients in the initial (freezing) stage of frozen shoulder syndrome (FSS) will describe the following:

- Insidious onset of vague, dull pain at the deltoid insertion
- Pain with shoulder movement
- Nagging pain at night, with sleep deprivation and the inability to sleep on the affected side
- Marked limitation of active and passive shoulder rotation, particularly external rotation
- The pain of FSS can be described as dull or aching. The pain leads to significant disability, affecting the activities of daily living, work, and leisure.
- During the second (frozen) stage, patients will complain of stiffness and severe loss of shoulder motion.
- The pain will be less pronounced. Patients will endorse the inability to reach over their head, to their side, and across their chest with the affected arm, or to scratch their back or put on a coat. The contralateral shoulder often becomes symptomatic years after FSS develops in the first shoulder.

Physical Examination

- The patient's posture should be observed while he or she is wearing a gown and sitting on a stool.
- On examination, the patient will have tenderness at the deltoid insertion and over the anterior capsule and posterior capsule with deep palpation.
- Compensatory scapulothroacic motion can create pain around the medial scapula. Forward flexion, abduction, and internal and external rotation of the shoulder should be assessed.
- Active and passive range of motion at the glenohumeral joint will be reduced, compared with the unaffected shoulder. Limitation of external rotation and abduction will be the most prominent findings.

Ancillary investigations & diagnosis

- Frozen shoulder syndrome (FSS) is a clinical diagnosis.
- Laboratory and imaging studies can be used to rule out other conditions and to confirm the

likelihood of the correct diagnosis. Accurate diagnosis is essential because the treatment for FSS differs from the treatment for other shoulder entities.

Laboratory Studies

- Laboratory studies rarely are required for the evaluation of adhesive capsulitis. However, if a predisposing medical condition that may be contributing to adhesive capsulitis is suggested, the following tests may be ordered:
- Complete blood cell count (CBC)
- Erythrocyte sedimentation rate (ESR)
- C-reactive protein (CRP)
- Blood glucose
- Hemoglobin A1c
- Thyroid-stimulating hormone (TSH)
- Free thyroxine index (FTI)
- Free thyroxine (FT4)

Imaging Studies

- Imaging studies are not indicated in the diagnosis of FSS.
- No imaging modality has been definitively shown to provide greater diagnostic value, due to the heterogeneity of techniques used, and the presence of potential confounding factors limits definitive conclusions from imaging study findings.
- Common findings in FSS are thickening of the coracohumeral and inferior glenohumeral ligaments.

Radiography

- Routine radiographs do not have a role in the diagnosis of FSS. However, plain films of the shoulder should be obtained in all cases to rule out any other pathologic process. These radiographs should include the following :
- Anteroposterior (AP) view of the glenohumeral joint in neutral rotation
- Supraspinatus outlet view
- Axillary lateral view (if possible)

Ultrasonography

- Ultrasound (regular and Doppler) for the diagnosis of FSS remains controversial.
- Overall, these studies have shown utility for measurement of acohumeral ligament thickness and presence of a hypoechoid region with increased vascularity in the rotator interval, with fibrovascular inflammatory tissue.

Arthrography

Arthrography, which involves intra-articular injection of diluted iodinated contrast medium, can aid in both the diagnosis and treatment of FSS.

Positron emission tomography/computed tomography

Duchstein et al suggest that 18-fluorodeoxyglucose positron emission tomography/computed tomography (^{18}F-FDG PET/CT) may have a role in diagnostically challenging cases.

Magnetic resonance imaging

- MRI is not initially indicated in cases of FSS. However, if the patient does not improve after a period of time (6 weeks to 3 months), then MRI is appropriate to rule out a possible rotator cuff tear or intra-articular pathology.

Histologic Findings

- Common arthroscopic findings in patients with adhesive capsulitis are the following:
- Proliferative synovitis
- Capsular and intra-articular subscapularis tendon thickening
- Fibrosis
- Chronic inflammatory cells

Lateral Epicondylitis (Tennis Elbow)

- Lateral epicondylitis (commonly referred to as "tennis elbow") is related to excessive wrist extension.
- It is the most common overuse syndrome.
- Patients typically report pain over the lateral elbow that worsens with activity and improves with rest.
- The symptoms tend to improve in 9-18 months; however, refractory cases may require surgery

Etiology

The causes of lateral epicondylitis include the following:

- Poor general conditioning leads to fatigue of the core and shoulder muscles, which puts an overemphasis on the extensor muscles of the forearm
- Improper training (eg, poor positioning when striking a tennis ball)
- Improper technique (eg, hitting a tennis ball late on the backhand)
- Poor or improper equipment (eg, a racquet that is strung too tightly)
- Scapular dyskinesis will lead to a compensatory increased load placed on the ipsilateral wrist extensors

Clinical Presentation

- The typical age of those affected is 40 to 50 years.
- Patients most typically report an insidious onset, but they will often relate a history of overuse without specific trauma.
- Symptom onset generally occurs 24-72 hours after repeated wrist extension activity.
- Delayed symptoms are probably due to microscopic tears in the tendon.
- The patient complains of pain over the lateral elbow that worsens with activity and improves with rest.
- The patient will also often describe aggravating conditions such as a backhand stroke in tennis or the overuse of a screwdriver.
- Pain may radiate down the posterior aspect of the forearm.
- Pain can vary from being mild (eg, with aggravating activities like tennis or the repeated use of a hand tool), or it can be such severe pain that simple activities like picking up and holding a coffee cup (ie, "coffee cup sign") will act as a trigger for the pain.

Physical Examination

The following may be noted on physical examination:

- Inspection: Very rarely does one notice swelling or ecchymosis.
- Palpation: Maximal tenderness is elicited 1-2 cm distal to the origin of the extensor carpi radialis brevis(ECRB) at the lateral epicondyle.
- Pain is increased with resisted wrist extension, with the wrist radially deviated and pronated and the elbow extended
- Increased pain is noted with resisted supination, gripping hand shaking.
- Always examine range of motion (ROM) of the shoulder, elbow, and wrist on the affected side.

Ancillary investigations -Diagnostic Studies

Laboratory studies

- Laboratory studies are typically not useful in the workup of lateral epicondylitis.

Imaging studies

- Consider plain film radiologic evaluation if the patient's symptoms persist despite adequate treatment or to evaluate for osteophytes, degenerative joint disease, or osteochondritis dissecans (OCD).
- Plain films may reveal osteophytes or calcifications along the lateral epicondyle.
- Consider magnetic resonance imaging (MRI), bone scanning, and/or computed tomography (CT) scanning to evaluate for OCD or stress fractures.
- Musculoskeletal ultrasonography is emerging as a useful modality to characterize areas of tendinosis, partial tearing, or calcifications and may assist with treatment options.

Other tests

A local anesthetic block may lead to symptom resolution and confirmation of the diagnosis.

Carpal Tunnel Syndrome

- Carpal tunnel syndrome (CTS) is a collection of characteristic symptoms and signs that occurs following compression of the median nerve within the carpal tunnel.
- It is characterised by numbness, paresthesias, and pain in the median nerve distribution. These symptoms may or may not be accompanied by objective changes in sensation

and strength of median-innervated structures in the hand.

Clinical presentation-Signs and symptoms

- Among the most common complaints, patients will reveal that their hands fall asleep or that things slip from their fingers without their noticing (loss of grip, dropping things); numbness and tingling also are commonly described.
- Such complaints should be localized to the palmar aspect of the first to the fourth fingers and the distal palm (ie, the sensory distribution of the median nerve at the wrist).
- The sensory symptoms above commonly are accompanied by an aching sensation over the ventral aspect of the wrist. This pain can radiate distally to the palm and fingers or, more commonly, extend proximally along the ventral forearm.
- Many patients with CTS also complain of a tight or swollen feeling in the hands and/or temperature changes (eg, hands being cold/ hot all the time).
- Many patients also report sensitivity to changes in temperature (particularly cold) and a difference in skin color. In rare cases, there are complaints of changes in sweating.

Ancillary investigations and diagnosis

- Electrophysiologic studies, including electromyography (EMG) and nerve conductions studies (NCS), are the first-line investigations in suggested CTS.
- Abnormalities on electrophysiologic testing, in association with specific symptoms and signs, are considered the criterion standard for CTS diagnosis. In addition, other neurologic diagnoses can be excluded with these test results.
- Electrophysiologic testing also can provide an accurate assessment of how severe the damage to the nerve is, thereby directing management and providing objective criteria for the determination of prognosis.
- Ultrasonography potentially can identify space-occupying lesions in and around the median nerve, confirm abnormalities in the median nerve (eg, increased cross-sectional area) that can be diagnostic of CTS, and help to guide steroid injections into the carpal tunnel.
- Magnetic resonance imaging (MRI) of the carpal tunnel is particularly useful preoperatively if a space-occupying lesion in the carpal tunnel is suggested.

Muscular Dystrophy

- Muscular dystrophy (MD) is a collective group of inherited noninflammatory but progressive muscle disorders without a central or peripheral nerve abnormality.
- The disease affects the muscles with definite fiber degeneration but without evidence of morphologic aberrations.
- Advances in molecular biology techniques illuminate the genetic basis underlying all types of MD: defects in the genetic code for dystrophin, a 427-kd skeletal muscle protein (Dp427).
- These defects result in the various manifestations commonly associated with MD, such as weakness and pseudohypertrophy.
- Dystrophin can also be found in cardiac smooth muscles and in the brain (accounting for the slight mental retardation associated with this disease).
- Minor variations notwithstanding, all types of MD have in common progressive muscle weakness that tends to occur in a proximal-to-distal direction, though there are some rare distal myopathies that cause predominantly distal weakness.
- The decreasing muscle strength in those who are affected may compromise the patient's ambulation potential and, eventually, cardiopulmonary function.
- Once wheelchair-bound, patients with MDs tend to develop worsening contractures, osteopenia (initially, followed by osteoporosis), and rapidly progressive scoliosis.

- The change in bone mineral density (BMD) can lead to fractures of the long bones or vertebrae, which in turn lead to more immobilization, disuse demineralization, and exacerbation of scoliosis.
- The goal of orthopedic management is, therefore, to preserve or prolong patients' ambulatory status for as long as possible. Etiology

Classification of types of muscular dystrophy

- The etiology of MD is an abnormality in the genetic code for specific muscle proteins.
- They all are classified according to the clinical phenotype, the pathology, and the mode of inheritance.
- The inheritance pattern includes the sex-linked, autosomal recessive, and autosomal dominant MDs. Within each group of heritable MDs several disorders exist. These are characterized by the clinical presentation and pathology.

Heritable MDs include the following:

- Sex-linked MDs - Duchenne, Becker, Emery-Dreifuss
- Autosomal dominant MDs - Facioscapulohumeral, distal, ocular, oculopharyngeal
- Autosomal recessive MD Limb-girdle form

Genetic defects and dystrophin

In the X-linked forms of MD, such as the Duchenne and Becker dystrophies, the defect is located on the short arm of the X chromosome. Dystrophin is distributed not only in skeletal muscle but also in smooth and cardiac muscles and in the brain. The large size of the dystrophin gene explains the ease at which spontaneous new mutations can occur, as in Duchenne MD. The large size also allows mistakes in protein synthesis to occur at multiple sites.

Clinical presentation History and Physical Examination

Three major time points for patients with Duchenne MD are as follows :

- When they begin to walk
- When they lose their ability to ambulate
- When they die

Phases of Duchenne muscular dystrophy

- The child's motor milestones may be at the upper limits of normal, or they may be slightly delayed.
- Some of the delays may be caused by inherent muscle weakness, but a component may stem from brain involvement.
- Other early features include a gait abnormality, which classically is a waddling, wide-based gait with hyperlordosis of the lumbar spine and toe walking. The waddle is due to weakness in the gluteus maximus and gluteus medius muscles and the patient's inability to support a single-leg stance. The child leans the body toward the other side to balance the center of gravity, and the motion is repeated with each step.
- Hip extensor weakness also results in a forward tilt of the pelvis, which translates to a hyperlordosis of the spine to maintain posture.
- Gradually, the child is observed to have increasingly noticeable difficulty with step taking.
- The child then begins having problems getting up from the sitting or supine position, and he or she can rise to an upright stance only by manifesting the Gowers sign.
- The Gowers sign is a classic physical examination finding in MD and results from weakness in the child's proximal hip muscles.
- Although the Gowers sign is a classic finding in Duchenne MD, it is by no means pathognomonic; other types of MD and disorders with proximal weakness may also cause this sign.
- While still ambulatory, the child may have minimal deformities, including iliopsoas or tendo Achillis tightness.
- The second important phase in Duchenne MD is the loss of ambulation.
- With the child's loss of ambulation, there is usually a rapidly progressive course of muscle or tendon contractures and scoliosis.

- Duchenne MD is a terminal disease in which death usually occurs by the third decade of life (mostly from cardiopulmonary compromise), despite steroid treatment.
- The respiratory failure can easily occur from the underlying progressive nocturnal hypoventilation and hypoxia or from an acute cardiac insufficiency.

Additional clinical findings in Duchenne muscular dystrophy

- Other clinical findings in Duchenne MD include absent deep tendon reflexes in the upper extremities and patella (though the tendo Achillis reflex remains intact even in the later stages of this disease), pain in the calves with activity, pseudohypertrophy of the calf and macroglossia .
- Cardiopulmonary involvement is present from the beginning of the disease stages, but the findings are not so clinically obvious. Electrocardiographic (ECG) tracings show right ventricular strain, tall R waves, deep Q waves, and inverted T waves.

Other types of muscular dystrophy

- Becker MD is similar to Duchenne MD, but because patients have some measure of functioning dystrophin, the manifestations of Becker MD occur later and are milder. Patients tend to live past the fourth or fifth decades.
- Emery-Dreifuss MD is an uncommon sex-linked dystrophy that presents with early contractures and cardiomyopathy in affected patients; the typical presentation involves tendo Achillis contractures, elbow flexion contractures, neck extension contractures, tightness of the lumbar paravertebral muscles, and cardiac abnormalities.
- Death may occur in the fourth or fifth decade as a result of first-degree atrioventricular (AV) block, a condition that is usually not present at the initial presentation of this disease.
- None of the autosomal dominant conditions significantly affect longevity.

Ancillary investigations-Laboratory Studies

- A creatine phosphokinase (CPK) determination is the most specific test for muscular dystrophy (MD).
- Elevated CPK levels are indicative of muscle disease. Because the concentration of CPK is not significant in red blood cells, CPK levels are not affected by hemolysis.
- CPK is not affected by liver dysfunction, as are the other tested enzymes (eg, transaminases, aldolase, lactate dehydrogenase).
- High CPK levels represent leakage of the enzyme from the muscle cells only. This change is not exactly correlated with the severity of the disease.
- All MDs result in some CPK elevation during the active phase of the disease. The finding of three elevated levels obtained 1 month apart is diagnostic for MD. Early in the disease process, CPK levels are 50-300 times greater than normal levels, but the levels tend to decrease as the muscle mass decreases. The CPK level is highest in Duchenne MD, with less elevation noted in Becker MD.

Enzyme levels that may be elevated but can be altered by liver dysfunction include the following:

- Transaminase levels
- Lactate dehydrogenase levels
- Aldolase levels
- PCR can be used to detect more than 98% of existing deletions, and it can be performed within 24 hours.

Imaging Studies

- Ultrasonography (US) is a relatively noninvasive technique that is used for screening patients with MD; this modality is rapidly replacing electromyography (EMG) in centers that have appropriately trained staff.

Other Tests

- Electrocardiography (ECG) is expected to show a right ventricular strain, tall R waves, deep Q waves, and inverted T waves.
- Pulmonary function tests (PFTs), including an

analysis of arterial blood gases, and a hematologic workup are necessary as part of the preoperative workup.

- EMG usually demonstrates short-duration, polyphasic, motor-unit action potentials with decreased amplitudes. It should be kept in mind that this finding is common with all myopathic processes and does not specifically identify MDs.

Biopsy

- Until the advent of molecular biology techniques, muscle biopsy was the definitive test for diagnosing and confirming muscular disease.
- Histologic changes depend on the stage of disease and the muscle selected. The optimal site for biopsy is the vastus lateralis, accessed via a small lateral thigh incision.

Histologic Findings

- Histologic specimens from muscle biopsy samples obtained early in the development of MD show only variations in muscle fiber sizes with focal areas of degenerating or regenerating fibers. In later stages of MD, the changes are more obvious, with marked variations in muscle fiber sizes, degeneration, and regeneration.

MCQs

1. आहारपरिणामान्तेभूयश्चलभतेबलम is clinical feature of
   a. व्यपेताहिक्का (Vyapeta Hikk
1. Carpal tunnel syndrome can be diagnosed by...
   a. No Visual examination by a doctor is required only Nerve conduction velocity test (NCV)
   b. Manujal Muscle Testing (MMT)
   c. Electrocardiography (ECG)
   d. Visual examination by a doctor, Nerve conduction velocity test (NCV) & Electromyogram (EMG)
2. Which of these is not a form of muscular dystrophy?
   a. Duchenne muscular dystrophy
   b. Facioscapulohumeral dystrophy
   c. Lunar dystrophy
   d. Myotonic dystrophy
3. Nerve affected in Carpal tunnel syndrome
   a. Median nerve
   b. Radial nerve
   c. Ulnar nerve
   d. Musculocutaneous nerve

Answer

1.	d	2.	c	3.	a

CHAPTER 45

Amavata

Introduction

Diagnosis of Rheumatoid arthritis. Indulgence in incompatible eatables and habits, lack of physical activity, or doing exercise after taking fatty eatables and those who have poor digestive capacity even normally also generate *āma* (improperly digested eatable) in the body. When a person of sedentary habits with a poor digestive faculty indulges in incompatible diet and regimen, or does physical exercise after taking fatty food the *āma* is formed. This *āma*, associating itself with *vāta*, moves quickly to the different seats of *kapha* in the body filling them and the *dhamanīs* (blood vessels) with waxy material. Thus the abnormal end product of digestion associated with *vāta*, *pitta* and *kapha* assuming different colours, blocks the tissue pores and passages with thick waxy material. It renders the patient weak in no time and produces a feeling of heaviness in the pericardial region. This substance named *āma* is the cause of so many distressing diseases. When provoked *āma* simultaneously afflicts the pelvic and shoulder, they girdle and make other joints of the body stiff. This condition has been identified as *āmavāta*. *(MN.* 25. 1-5*)*

Hetu of Āmavāta (Etiological factors of Āmavāta)

विरुद्धाहारचेष्टस्य मन्दाग्नेर्निश्चलस्य च।
स्निग्धं भुक्तवतो ह्यन्नं व्यायामं कुर्वतस्तथा॥
(M.Ni 24/1)

For the first time hetu of *Āmavāta* was first described by *Madhukoṣakara* in *Mādhava Nidāna* are as follows-

Disagreement dietetics and drinks; hindered activities or behaviour, slowness or inactive in the digestive mechanism. unchangeable or sedentary habits; consumption of sticky food followed by exercise.

Pūrvarūpa (Prodromal Symptoms)

The only text which describes the premonitory signs and symptoms is *Vaṅgasena Saṁhitā* namely:- *Śirā rūja* (Cephalgia) and *Gātra rūja* (Bodyache).

Samprāpti of Āmavāta

वायुना प्रेरितो ह्यामः श्लेष्मस्थानं प्रधावति।
तेनात्यर्थं विदग्धोऽसौ धमनीः प्रतिपद्यते॥
वातपित्तकफैर्भूयो दूषितः सोऽन्नजो रसः।
स्त्रोतोंस्यभिष्यन्दयति नानावर्णोऽतिपिच्छिलः॥
जनयत्याशु दौर्बल्यं गौरवं हृदयस्य च।
व्याधीनामाश्रयो ह्येष आमसंज्ञोऽतिदारुणः॥
युगपत्कुपितावन्तस्त्रिकसन्धिप्रवेशकौ।
स्तब्धं च कुरुतो गात्रमामवातः स उच्यते॥
(M.Ni 24/2-5)

Due to ingestion of the etiological factors, which mentioned earlier, disturbs the normal functions of agni as a result food not properly digested, yields immature *Rasa* in the *Āmāśaya* and this undergoes fermentation and/or putrefaction because of its retention for longer duration, manifests *Āma*. Then it is absorbed in the system and it is taken up by the aggravated and vitiated *Vāyu* specially to the *Kaphasthānās* mainly *Āmāśaya*, *Sandhi*, *Ūras*, *Kaṇṭha* etc. and the same undergoes in the above mentioned sites and enters the *Dhamani* by circulation with the help of *Vāta*. Here it amalgamates with the existing *Doṣās* and vitiated further in excess and produces distress in the body channels due to its excessive unctuous nature and some other different qualities of *Āmarasa*. This leads to general weakness and heaviness in the precordial region, and this *Āmarasa* is responsible for the genesis of symptom complex, that's why this disease is considered challenging and difficult to treat. So both *Āma* and *Vāta* aggravates simultaneously and enters *Koṣṭha*, *Trika* and *Sandhi* and ultimately leads to *Stabdhatā* in the body. This is called *Āmavāta*.

Nidān Sevana (Consumption of Etiological factors)

↓

Aggravation of *Kapha doṣa*
↓
Enters the *Āmāśaya*
↓
Disturbs the *Agni*
↓
Yields immature *Rasa* in the *Āmāśaya* i.e. *Āma*
↓
Undergoes fermentation and/or putrefaction because of its retention for longer duration
↓
Āma absorbed in the system
↓
It is taken up by the aggravated and vitiated *Vāyu* specially to the *Kaphasthānās* mainly *Āmāśaya*, *Sandhi*, *Ūras*, *Kaṇṭha* etc.
↓
Enters the *Dhamani* by circulation with the help of *Vāta*
↓
Amalgamate with the existing *Doṣās* there
↓
Vitiates further in excess and produces distress in the body channels due to its excessive unctuous nature and some other different qualities of *Āmarasa*.
↓
This leads to general weakness and heaviness in the precordial region
↓
Āmarasa is responsible for the genesis of symptom complex
↓
That's why this disease is considered challenging and difficult to treat
↓
So both *Āma* and *Vāta* aggravates simultaneously and enters *Koṣṭha*, *Trika* and ultimately leads to *Stabdhatā* in the body
↓
Āmavāta.

आमवात संप्राप्ति घटक (Samprāpti Ghaṭaka of Āmavāta)

Doṣa : *Vāta →Vyāna, Samāna, Prāṇa*
Pitta → Pācaka
Kapha → Śleṣaka

Dūṣya : *Dhātu → Rasa, Meda, Majjā*
Upadhātu → Sirā, Snāyu
Śārīrika Mala → Puriṣa
Dhātu Mala → Kapha

Agni : *Jaṭharāgni* and *Dhātvāgni*

Agniduṣṭi : *Mandāgni*

Āma : *Āgnijanya*

Srotas : *Annavaha, Rasavaha, Asthivaha*

Srotoduṣṭi : *Saṅga*

Udbhavasthāna : *Āmāśaya, Pakvāśaya*

Sancārasthāna : *Annavaha srotas, Dhamani, Kaphasthāna*

Adhiṣṭhāna : *Kaphasthāna* like *Sandhi*, *Āmāśaya* etc.

Vyaktasthāna : *Kaphasthāna* i.e., *Sandhi*, *Āmāśaya*, *Ūras* etc.

Svabhāva : *Kaṣṭasādhya*

Roga Mārga : *Madhyama*

Cardinal Signs and Symptoms of Āmavāta

अङ्गमर्दोऽरुचिस्तृष्णा ह्यालस्यं गौरवं ज्वरः ।
अपाकः शूनताऽङ्गानामामवातस्य लक्षणम् ॥
(M.Ni 24/6)

General sings and symptoms of *Āmavāta* are bodyache, anorexia, thirst, heaviness, fever, indigestion and numbness

Signs and symptoms of advanced Āmavāta

स कष्टः सर्वरोगाणां यदा प्रकुपितो भवेत् ।
हस्तपादशिरोगुल्फत्रिकजानूरुसन्धिषु ॥
करोति सरुजं शोथं यत्र दोषः प्रपद्यते ।
स देशो रुज्यतेऽत्यर्थं व्याविद्ध इव वृश्चिकैः ॥
जनयेत्सोऽग्निदौर्बल्यं प्रसेकारुचिगौरवम् ।
उत्साहहानिं वैरस्यं दाहं च बहुमूत्रताम् ॥
कुक्षौ कङ्गिनतां शूलं तथा निद्राविपर्ययम् ।
तृट्छर्दिभ्रममूर्च्छाश्च हृद्ग्रहं विड्विबद्धताम् ।
जाड्यान्त्रकूजमानाहं कष्टाश्चन्यानुपद्रवान् ॥
(M.Ni 24/7-10)

Painful swelling of the joints i.e. hand, feet, ankle, knee, hip and spinal column, nature of pain is like that of scorpion sting, stiffness, hindered digestive mechanism, excessive salivation, anorexia, heaviness, lack of enthusiasm, distaste in the mouth, burning sensation, excessive urination, hardness and pain in the abdomen, disturbed sleep, thirst, vertigo, fainting, stiffness in precordium, constipation, stiffness, intestinal sounds, distension of abdomen, severe difficulties with complications.

Viśiṣṭa Lakṣaṇās (special cardinal signs and symptoms)

पित्तात्सदाहरागं च सशूलं पवनानुगम्।
स्तिमितं गुरुकण्डूं च कफदुष्टं तमादिशेत्॥
(M.Ni 24/11)

Totally 7 types of *Āmavāta* mentioned according to dominancy of *doṣās* in *Mādhava Nidāna* & *Yogaratnākara*.

1. *Vātaja Āmavāta* - Characterised by agonizing pain in the affected parts.
2. *Pittaja Āmavāta* - Characterised by burning and redness of the affected joints.
3. *Ślaiṣmika Āmavāta* - Characterised by rigid, heaviness, itching of the affected joints.
4. *Vāta-Pittaja Āmavāta* - Combination of vata and *Pittaja Āmavata* signs & symptoms
5. *Pitta-Kaphaja Āmavāta* - Combination of signs & symptoms of *Pittaja & Kaphaja Āmavata.*
6. *Vāta-Kaphaja Āmavata* - Manifestation of both *Vataja & Kaphaja Āmavata Symptoms*
7. *Sannipātaja Āmavata* - Combination of all signs and symptoms of three *Doṣas* along with swelling all over the body.

Sādhyāsādhyatva (prognosis)

एकदोषानुगः साध्यो द्विदोषो याप्य उच्यते।
सर्वदेहचरः शोथः कृच्छ्रः सान्निपातिकः॥
(M.Ni 24/12)

Various authors like *Mādhava Nidāna, Vaṅgasena Saṁhitā, Bhāvaprakāśa, Yogaratnākara, Gadanigraha* and *Śabdakalpa druma* etc. have explained the prognosis of *āmavāta* are as follows :

Dominancy of single *doṣa* in *āmavāta* indicates disease become easily curable. Dominancy of two *doṣa* in *āmavāta* indicates manageable condition and dominancy of all the three *doṣās* indicates difficult to cure.

उपशय/पथ्य (Upaśaya/Pathya)

A patient of *Āmavāta* who suffers morbid thirst should drink water which has been boiled along with *Pippalī, Pippalīmūla, Cavya, Citraka* and dried ginger. One should consume brinjal cooked in *Kānji* of *Sauvīra* and boiled fruits of *Tikta.* An *Āmavāta* patient's diet should include leaves of *Vāstuka*, leaves of *Neem*, leaves of *Punarnavā, Paṭola*, leaves of *Gokṣura*, leaves of *Varuṇa* and *Kāravellaka*, barley, *Kodrava*, old varieties of *Śāli* and *Ṣaṣṭhi* rice, meat of *Lāva* bird treated with buttermilk (*Takra*), pulse soup of *Kulattha* beans, peas as well as gram and the like. The patient should also take fomentation through warm sand particles packed in a piece of cotton. (B.R. 29/2-8)

The following measures are beneficial for a patient of *Āmavāta*: *Svedana* therapy of *Rūkṣa* type, *Laṅgana* (fasting), *Snehapāna*, *Basti* therapy, *Lepa* and the like. The patient should take the following eatables: old rice, *Kulattha* pulse, old liquor, meat soup of wild animals, buttermilk, *Varṣābhū, Eraṇḍa* oil, garlic, warm water, *Paṭola* leaves, *Kāravellaka*, brinjal, drumstick, ginger, cow urine and such herbs as *Gokṣura, Vṛiddhdāraka* and *Bhallātaka*. The bitter and sour edibles and those meals, which are easily digestible, are useful for the patient. (B.R. 29/232-234)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures are harmful for a patient of *Āmavāta*: facing easterly winds, eating incompatible meals, holding the force of urine and stools, excessive walking and the like. The patient should avoid the following eatables: curd, buttermilk, fish, raw sugar, milk, *Upodikā* leaves, *Urada*, contaminated water and the like. (B.R. 29/235-236)

The *Abhiṣyandaka* eatables, the edibles that are heavy to digest and the food containing oily substances should be purposely avoided by the patient of *Āmavāta*. (B.R. 29/237).

MCQs

1. *Vyaviddha iva vrischikai* is the feature of which disease
   a. *Amavata* b. *Vatarakta*
   c. *Udavarta* d. *Urustambha*
2. *Bahumutrata* seen in
   a. *Amavata Samanyalakshana*
   b. *Pravriddha Amavata lakshana*
   c. *Kaphaja Amavatalakshana*
   d. *Vataja Amavatalakshana*
3. Prognosis of *Dwidoshaja Amavata*
   a. *Sadya* b. *Krichrasadya*
   c. *Yapya* d. *Anupakrama*
4. Which among the following is not the *Samanyalakshana* of *Amavata*
   a. *Angamarda* b. *Hrillasa*
   c. *Shoonanga*
   d. *Daha toda spurana paka*
5. जाड्यान्त्रकूजमानाहंकष्टांश्चान्यानुपद्रवान् is about
   a. *Amavata Samanyalakshana*
   b. *Pravridha Amavatalakshana*
   c. *Kaphaja Amavatalakshana*
   d. *Gulmaasadyalakshana*
6. Which among the following is not the feature of *Kaphapradhana Amavata*
   a. *Sthimita* b. *Sopha*
   c. *Guru* d. *Kandu*

Answer

1.	a	2.	b	3.	c
4.	d	5.	b	6.	b

CHAPTER

46

Vata-Rakta

Introduction

Generally in persons who have tender health, who indulge in wrong diet and regimen, who are obese and who lead a leisurely life, *vāyu* and *rakta* get aggravated because of the following factors: intake of food which is saline, sour, pungent and alkaline in taste; intake of ununctuous and hot food; intake of food before the previous meal is digested; intake of meat of aquatic and marshy land animals; intake of oil cake, raddish, *kulattha*, *māṣa*, *niṣpāva*, leafy vegetables, *palala* sugarcane juice and its preparations, curd *aranāla* (a sour drink prepared by acid-fermentation), *sauvīra* (vinegar), *śūkta* (another type of vinegar), butter milk, *surā* (alcoholic drink) and *āsava* (a type of alcoholic drink); Intake of mutually contradictory food; *Adhyasana* (intake of second meal immediately after first meal); and anger, sleep during day time and remaining awake at night. Persons who indulge in eating while riding an elephant, a horse or a camel, and persons who indulge in eating *vidāhi* food (which causes burning sensation), his entire blood gets vitiated and gets accumulated in the legs. This gets further vitiated by the aggravated *vāyu*. Subsequently, *vāta-rakta* (gout) is manifested.

Excessive sweating or absence of sweating; appearance of grayish black patches on the body; lack of touch sensation; excessive pain by injury; looseness of joints; laziness, prostration and appearance of boils; excruciating pain, cramps, breaking pain, heaviness and numbness in knee joints, calf region, thighs, lumber region, shoulder region and other joints of upper limbs, lower limbs and different parts of the body; itching and pain in joints which often appear and disappear; and discoloration of the skin and appearance of circular patches over it.

The patient of *vāta rakta* with the following complications is incurable: Sleeplessness, anorexia, dyspnoea, *māmsa kotha* (sloughing of muscle tissue) and stiffness of head; fainting, excessive prostration, excessive pain, morbid thirst, fever, perspirationness, trembling, hiccup, *pāṅgulya* (inability to walk), erysipelas, suppuration, pinching pain, giddiness and fatigue. Curvature and deformity of fingers and toes; eruptions, burning sensation, stiffness of vital organs and tumours; and unconsciousness. The patient suffering from *vāta rakta* for more than one year is also incurable. If the patient has only a few of the complications, then the conditon is *yāpya* (palliable). If, however, he is free from complications, then he is curable. *Vāta rakta* is of two types: viz. *uttāna* (superficial) and *avagāḍha* (deep-seated). The former afflict the skin and the muscle tissue, while the later is deep seated. For both the type of *vāta rakta*, blood-letting therapy is administered with the help of *śṛṅga* (horn of animals), *sūcī* (prickling by needles), *alābu* and leeches. If the *vāta rakta* is of shifting type then blood-letting is performed by venesection or *pracchanna* (scraching) if the disease is associated with fatigue and if as a result of aggravated *vāyu*, there is ununctuousness in the body, then also blood-letting is performed by venesection. Gout; Acute pain and inflammation of joints which begins from the small toe or finger, and subsequently gradually spreads to the other joints of the body, extreme tenderness and burning sensation in the involved joints, excessive sweating, rigidity, numbness, discolouration of the skin on the affected joint, are the general signs and symptoms of *Vātarakta*. Ban. *bāt rakt*, Guj. gthiyo, Hin. *gṭhiā*, Kan. *vātrakt*, Mal. *vātraktam*, Mar. *vāt rakt*, Ori. *ambilipitī*, Tam. *vāt raktam*. (*CS*. *Ci*. 29. 5 -7; *SS*. *Ci*. 5. 5).

वातरक्त हेतु (Vāta-Rakta Hetu)

लवणाम्लकटुक्षारस्निग्धोष्णाजीर्णभोजनैः ।
क्लिन्नशुष्काम्बुजानूपमांसपिण्याकमूलकैः ॥
कुलत्थमाषनिष्पावशाकादिपललेक्षुभिः ।
दध्यारनालसौवीरशुक्ततक्रसुरासवैः ॥

विरुद्धाध्यशनक्रोधदिवास्वप्नप्रजागरैः ।
प्रायशः सुकुमाराणां मिष्टान्नसुखभोजिनाम् ॥
अचङ्क्रमणशीलानां कुप्यते वातशोणितम् ।
अभिघातादशुद्ध्या च प्रदुष्टे शोणिते नृणाम् ॥
कषायकटुतिक्ताल्परूक्षाहारादभोजनात् ।
हयोष्ट्रयानयानाम्बुक्रीडाप्लवनलङ्घनैः ॥
उष्णे चात्यध्वगमनाद्व्यवायाद्वेगनिग्रहात् ।

C.S.Ci 29/5-10

प्रायशः सुकुमाराणां मिथ्याऽऽहारविहारिणाम् ।
शोकाच्च प्रमदामद्यव्यायामैश्चातिपीडनात् ॥
ऋतुसात्म्यविपर्यासात् स्नेहादीनां च विभ्रमात् ।
अव्यवाये तथा स्थूले वातरक्तं प्रकुप्यति ॥
हस्त्यश्वोष्ट्रैर्गच्छतोऽन्यैश्च वायुः कोपं यातः कारणैः सेवितैः स्वैः।
तीक्ष्णोष्णाम्लक्षारशाकादिभोज्यैः सन्तापाद्यैर्भूयसा सेवितैश्च ॥

S.S.Ni 1/40-42

The following etiological factors mentioned for the manifestation of *Vāta-rakta*.

Rakta Prakopaka Nidāna : Regular consumption of saline, sour, pungent, alkaline, unctuous, hot and uncooked food; regular intake of putrified or dry substances; meat of aquatic or marshy land inhabiting animal; oil cakes; radish; horsegram; blackgram; *Nişpāva;* other vegetables (leguminous); sesamum paste; sugarcane; curd; sour gruel; *sauvīra;* (sour preparation of dehusked barely, etc.), *Sukta* (vinegar), butter milk, alcohol & wine; intake of mutually contradictory food; eating during indigestion, anger; sleeping during daytime and awakening in night; delicate people, who indulge excess sweet preparations, eating abundantly and adopting sedentary life style; due to injury and avoidance of the bio-purification therapies when needed.

Vāta Prakopaka Nidāna

Excessive intake of astringent, pungent, bitter and unctuous substances; less intake of food or fasting; riding over horses and camels or on carts carried by them; resorting to aquatic games like swimming, jumping; excess traveling in hot season by bare foot; sexual intercourse and suppression of natural urges.

Suśruta included some other factors like one who is suffering from chronic diseases; long walk; intake of wine in excess; excessive physical activities; one who indulge in food & activities opposite to the season; who obstains from sexual intercourse; those who are obese etc.

वातरक्त संप्राप्ति (Vāta-Rakta Samprāpti)

वायुर्विवृद्धो वृद्धेन रक्तेनावारितः पथि ॥
कृत्स्नं संदूषयेद्रक्तं तज्ज्ञेयं वातशोणितम् ।
खुडं वातबलासाख्यमाढ्यवातं च नामभिः ॥

C.S.Ci 29/10-11

क्षिप्रं रक्तं दुष्टिमायाति तच्च वायोर्मार्गं संरुणद्ध्याशु यातः ।
क्रुद्धोऽत्यर्थं मार्गरोधात् स वायुरत्युद्रिक्तं दूषयेद्रक्तमाशु ॥
तत् संपृक्तं वायुना दूषितेन तत्प्राबल्यादुच्यते वातरक्तम् ।
तद्वत् पित्तं दूषितेनासृजाऽऽक्तं श्लेष्मा दुष्टो दूषितेनासृजाऽऽक्तः ॥

S.S.Ni. 1/43-44

Due to above etiological factors *Vāta* and *Rakta* excacerbate simultaneously. Aggravated *Vāta* having been obstructed in its passage by aggravated blood vitiates the entire blood. This is known as *Vāta-Rakta*. It is also known by synonyms like *Khuḍa, Vāta-Balāsa* and *Āḍhya-Vāta*. When aggravated *Pitta* combining with vitiated blood is known as *Pitta-Rakta* and when excacerbated *Kapha* combining with vitiated blood is known as *Kapha-Rakta.*

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Excacerbation of *Vāta* and *Rakta* simultaneously
↓
Obstruction in the *Lohitavaha Srotas* for *vāta* movement
↓
Vitiates the whole *Rakta*
↓
Vāta-Rakta

(It is caused by the vitiation of *Rakta* by *Vāta*)

Pathogenesis of Pitta-Rakta

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Pittādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Excacerbation of *Pitta* and *Rakta* simultaneously
↓
Obstruction in the *Lohitavaha Srotas* for *Pitta* movement
↓
Vitiates the whole *Rakta*
↓
Pitta-Rakta
(It is caused by the vitiation of *Rakta* by *Pitta)*

Pathogenesis of Kapha-Rakta

Nidān Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kaphādi doṣās*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Excacerbation of *Kapha* and *Rakta* simultaneously
↓
Obstruction in the *Lohitavaha Srotas* for *Kapha* movement
↓
Vitiates the whole *Kapha*
↓
Kapha-Rakta
(It is caused by the vitiation of *Rakta* by *Kapha)*

वातरक्त संप्राप्ति घटक (Samprāpti Ghaṭaka of Vāta-Rakta)

Doṣa : *Vāta pradhāna tridoṣaja*
Vāta → Vyāna
Pitta → Pācaka
Kapha → Śleṣaka

Dūṣya : *Dhātu → Uttāna Vāta-Rakta→ Rakta, Rasa, Māmsa*
Gambhīra Vāta-Rakta→ Rakta, Rasa, Māmsa, Meda, Asthi & Majjā.
Upadhātu → Sira, Kaṇḍarā, Sandhi, Snāyu, Tvacā
Śārīrika mala→Mūtra, Puriṣa
Dhātumala → Kapha, Sveda & Pitta

Agni : *Jaṭharāgnijanya, Dhātvāgnijanya*

Agniduṣṭi : *Mandāgni*

Āma : *Agnijanya*

Srotas : *Vātavaha, Rasavaha, Rakta-vaha* initially later all *Dhātu vaha srotas*

Srotoduṣṭi : *Saṅga*

Adhiṣṭhāna : *Jānu, Jaṅgā, Uru, Kaṭi, Amsa, Dhamani, Hastapādānga Sandhi*

Udbhavasthāna : *Pakvāśayottha, Āmāśayottha*

Vyaktasthāna : *Jānu, Jaṅgā, Uru, Kaṭi, Amsa, Dhamani, Hastapādānga Sandhi*

Sancārasthāna : *Rasa, Raktavāhinyaḥ*

Svabhāva : *Āśukāri*

Prabhāva : *Ekadoṣaja→Sādhya*
Dvidoṣaja→Yāpya
Tridoṣaja→Asādya

Roga Mārga : *Madhyama*

Roga bheda : 1. *Uttāna*
2. *Gambhīra*
3. *Ubhayāśṛaya*

वातरक्त मूल स्थान (Vāta-Rakta Mūla Sthāna)

तस्य स्थानं करौ पादावङ्गुल्यः सर्वसन्धयः।
कृत्वाऽऽदौ हस्तपादे तु मूलं देहे विधावति॥
सौक्ष्म्यात् सर्वसरत्वाच्च पवनस्यासृजस्तथा।

तदद्रवत्वात् सरत्वाच्च देहं गच्छन् सिरायनैः ॥
पर्वस्वभिहतं क्षुब्धं वक्रत्वादवतिष्ठते ।
स्थितं पित्तादिसंसृष्टं तास्ताः सृजति वेदनाः ॥
करोति दुःखं तेष्वेव तस्मात् प्रायेण सन्धिषु ।
भवन्ति वेदनास्तास्ता अत्यर्थं दुःसहा नृणाम् ॥
C.S.Ci 29/12-15

Vāta-Rakta manifest in hands, feet, fingers and all joints. Initially hand and feet are afflicted and later it spreads to all over the body. Due to subtleness and pervasiveness of *Vāta* and liquidity and dispersibility of *rakta* it circulates all over the body through blood vessels, get obstructed in the joints due to zigzagway, which makes them further aggravated and gets lodged there. Further in the joints, in combination with *Pittādi Doṣās* causes respective afflictions and produces different types of pain in the joints, which is very severe and patients suffers from such pain becomes unbearable.

पूवरूप (Pūrvarupa)

स्वेदोऽत्यर्थं न वा कार्ष्ण्यं स्पर्शाज्ञत्वं क्षतेऽतिरुक् ।
सन्धिशैथिल्यमालस्यं सदनं पिडकोद्गमः ॥
जानुजङ्घोरुकट्यंसहस्तपादाङ्गसन्धिषु ।
निस्तोदः स्फुरणं भेदो गुरुत्वं सुप्तिरेव च ॥
कण्डूः संधिषु रुग्भूत्वा भूत्वा नश्यति चासकृत् ।
वैवर्ण्यं मण्डलोत्पत्तिर्वातासृक्पूर्वलक्षणम् ॥
C.S.Ci 29/16-18

तस्य पूर्वरूपाणि तोददाहकण्डूशोफस्तम्भत्वक्पारुष्य-सिरास्नायुधमनीस्पन्दनसक्थिदौर्बल्यानि श्यावारुणमण्डलोत्पत्तिश्चाकस्मात् पाणिपादतलाङ्गुलिगुल्फमणिबन्धप्रभृतिषु तत्राप्रतिकारिणोऽपचारिणश्च रोगो व्यक्ततरः, तस्य लक्षणमुक्तं तत्राप्रतिकारिणो वैकल्यं भवति ॥ *C.S.Ci 5/4*

The premonitory signs and symptoms of the *Vāta-Rakta* are as follows : Excess or absence of perspiration; blackishness; anesthesia; severe pain if there is injury; looseness of joints; lassitude; malaise; skin eruption; pricking pain, quivering, tearing, heaviness and numbness in the knees, calf region, thighs, umbilical region, shoulders, hands, feet and joints in the body; itching frequently; pain subsides & reappears; discolouration in the skin and appearance of circular patches over the body; rigidity; roughness of the skin; throbbing of veins, ligaments and arteries; debility of the leg; appearance of bluish red patches without any aparant cause developing in the palms, soles, fingers, ankle, wrist etc.; at this stage if treatment is not initiated or if the person indulges the causative factors leading to complete manifestation of the disease. If not treated properly it produces deformities.

Classification of Vāta-Rakta

उत्तानमथ गम्भीरं द्विविधं तत् प्रचक्षते ।
त्वङ्मांसाश्रयमुत्तानं गम्भीरं त्वन्तराश्रयम् ॥
C.S.Ci 29/19

It is classified into two varieties i:e *Uttāna* & *Gambhīra. Uttāna* is situated superficially in the skin along with muscle tissues. *Grambhīra* is located in deeper tissue of the body.

तत्र वातेऽधिके वा स्याद्रक्ते पित्ते कफेऽपि वा ।
संसृष्टेषु समस्तेषु यच्च तच्छृणु लक्षणम् ॥
C.S.Ci 29/24

Classification of *Vāta-Rakta* is based on predominance of *Doṣās*

a) *Vāta predominant Vāta-Rakta*
b) *Pitta predominant Vāta-Rakta*
c) *Kapha predominant Vāta-Rakta*
d) *Dvandva Doṣa predominant Vāta-Rakta*
e) *Tridoṣaja predominant Vāta-Rakta*

According to Suśruta

द्विविधं वातशोणितमुत्तानमवगाढं चेत्येके भाषन्ते । तत्तु न सम्यक्, तद्धि कुष्ठवदुत्तानं भूत्वा कालान्तरेणावगाढीभवति, तस्मान्न द्विविधम् ॥ *S.S.Ci 5/3*

Some people classify *Vāta-rakta* into two types *Uttāna* and *Gambhīra* but this is not correct because it develops first as *Uttāna* and later becomes *Gambhīra*, just like *Kuṣṭha,* that is why it should not be classified into two types.

Common Signs and Symptoms of Vāta-Rakta

स्पर्शोद्विग्नो तोदभेदप्रशोषस्वापोपेतौ वातरक्तेन पादौ ।
पित्तासृग्भ्यामुग्रदाहौ भवेतामत्यर्थोष्णौ रक्तशोफौ मृदु च ॥
कण्डूमन्तौ श्वेतशीतौ सशोफौ पीनस्तब्धौ श्लेष्मदुष्टे तु रक्ते ।
सर्वैर्दुष्टे शोणिते चापि दोषाः स्वं स्वं रूपं पादयोर्दर्शयन्ति ॥
S.S.Ni 1/45-46

Patient will not tolerate touch in feet region and develops severe pain i:e either pricking or cutting in the feet; profound dryness; wasting; loss of sensation.

With the association of *Pitta* & *Rakta* produces severe burning sensation, profound heat, redness and soft swelling in the feet.

With the association of *Kapha* & *Rakta* causes swelling, itching, whitishness, coldish touch, bulged up and immovable. With the association of all the *Doṣās* with *Rakta* causes respective signs & symptoms of all the three *Doṣās* in the legs.

पादयोर्मूलमास्थाय कदाचिद्धस्तयोरपि ।
आखोर्विषमिव क्रुद्धं तद्देहमनुसर्पति ॥

S.S.Ni 1/48

Generally it starts from feet but some times it may start from hands, getting increased, spreads to entire body like rat poison.

Specific signs & symptoms of Vāta-Rakta

उत्तान वातरक्त (Uttāna Vātarakta)

कण्डूदाहरुगायामतोदस्फुरणकुञ्चनैः ।
अन्विता श्यावरक्ता त्वग्बाह्ये ताम्रा तथेष्यते ॥

C.S.Ci 29/20

Uttāna Vātarakta is characterised by itching, burning sensation, pain, stretching, piercing pain, quivering and contraction. The skin colour changed to brownish black, red or coppery in colour.

गम्भीर वातरक्त (Gambhīra Vātarakta)

गम्भीरे श्वयथुः स्तब्धः कठिनोऽन्तर्भृशार्तिमान् ।
श्यावस्ताम्रोऽथवा दाहतोदस्फुरणपाकवान् ॥
रुग्विदाहान्वितोऽभीक्ष्णं वायुः सन्ध्यस्थिमज्जसु ।
छिन्दन्निव च रत्यन्तर्वक्रीकुर्वंश्च वेगवान् ॥
करोति खञ्जं पङ्गुं वा शरीरे सर्वतश्चरन् ।

C.S.Ci 29/21-23

Gambhīra Vātarakta is characterised by swelling, stiffness, hardness, severe pain in interior part of the body, blackish brown or coppery discolouration, burning sensation, pricking pain, quivering and inflammation. Aggravated *Vāta* moves with high speed brings pain & burning sensation in the joints, bones and bone marrow as if cutting and making them crooked produces limping or lameness while moving all over the body.

उभयाश्रय (Ubhayāśraya)

All the signs & symptoms of *Uttāna* & *Gambhīra* are manifested.

वात प्रधान वातरक्त (Vāta Pradhāna Vāta-Rakta)

विशेषतः सिरायामशूलस्फुरणतोदनम् ।
शोथस्य कार्ष्ण्यं रौक्ष्यं च श्यावतावृद्धिहानयः ॥
धमन्यङ्गुलिसन्धीनां सङ्कोचोऽङ्गग्रहोऽतिरुक् ।
कुञ्चनस्तम्भने शीतप्रद्वेषश्चानिलेऽधिके ॥

C.S.Ci 29/25-26

It is characterised by dilatation of vessels, colic pain, quivering, piercing pain, blackishness, roughness and brownishness of swelling; along with its increase & decrease, contraction in the blood vessels, fingers and joints; stiffness in the extremities, intense pain; contraction & stiffness in the involved parts and aversion towards cold substances.

रक्त प्रधान वात-रक्त (Rakta pradhāna Vāta-Rakta)

श्वयथुर्भृशरुक् तोदस्ताम्रश्चिमिचिमायते ।
स्निग्धरुक्षैः शमं नैति कण्डूक्लेदान्वितोऽसृजि ॥

C.S.Ci 29/27

Rakta pradhāna Vātarakta is characterised by swelling with severe distress, pricking pain, coppery discolouration, tingling sensation, do not subside by either unctuous or rough applications; itching and sloughing.

पित्तप्रधान वातरक्त (Pitta Pradhāna Vāta-Rakta)

विदाहो वेदना मूर्च्छा स्वेदस्तृष्णा मदो भ्रमः ।
रागः पाकश्च भेदश्च शोषश्चोक्तानि पैत्तिके ॥

C.S.Ci 29/28

It is characterised by burning sensation, pain, fainting, sweating, morbid thirst, narcosis & giddiness, redness, inflammation, tearing and wasting of the extremities.

कफप्रधान वातरक्त (Kapha Pradhāna Vāta-Rakta)

स्तैमित्यं गौरवं स्नेहः सुप्तिर्मन्दा च रुक् कफे ।

C.S.Ci 29/29

It is characterised by feeling of wetness, heaviness, unctuousness, numbness associated with mild pain.

द्विदोष या त्रिदोष प्रधान वातरक्त (Dvidoṣa or Tridoṣa Pradhāna Vātarakta)

Vātarakta caused either by two *Doṣa*

dominancy or three *Doṣa* dominancy should be known by combined etiology and symptoms.

साध्यासाध्यता (Sādhyasādhyatā)

एकदोषानुगं साध्यं नवं, याप्यं द्विदोषजम्।
त्रिदोषजमसाध्यं स्याद्यस्य च स्युरुपद्रवाः॥

C.S.Ci 29/30

If *Vātarakta* caused by predominance of single *Doṣa* & newly manifested is curable.

If *Vātarakta* caused by dominancy of two *Doṣās* is palliable. If *Vātarakta* caused by dominancy of all the three *doṣās* along with complications is incurable.

उपद्रव (Upadrava)

अस्वप्नारोचकश्वासमांसकोथशिरोग्रहाः।
मूर्च्छायमदरुक्तृष्णाज्वरमोहप्रवेपकाः॥
हिक्कापाङ्गुल्यवीसर्पपाकतोदभ्रमक्लमाः।
अङ्गुलीवक्रता स्फोटा दाहमर्मग्रहार्बुदाः॥
एतैरुपद्रवैर्वर्ज्यं मोहेनैकेन वाऽपि यत्।
संप्रस्त्रावि विवर्णं च स्तब्धमर्बुदकृच्च यत्॥
वर्जयेच्चैव संकोचकरमिन्द्रियतापनम्।
अकृत्स्नोपद्रवं याप्यं साध्यं स्यान्निरुपद्रवम्॥

C.S.Ci 29/31-34

The following complications observed in *Vātarakta* patients : Sleeplessness, anorexia, dyspnoea, sloughing, stiffness in head, fainting, narcosis, distress, morbid thirst, fever, unconsciousness, trembling, hiccup, lameness, erysipelas, inflammation, pricking pain, giddiness, exhaustion, curvature of fingers & toes, blisters, burning sensation, afflictions in vital parts and tumours. Such patients should not be managed or even one complication i:e unconsciousness is enough to judge incurable nature of the disease.

If *Vātarakta* associated with profuse discharge having abnormal complexion of the skin, stiffness, tumor, contraction and afflictions of the senses, such patients should not be treated. Patients suffering from few complications may be palliable and absence of complications is curable.

उपशय/पथ्य (Upaśaya/Pathya)

Auṣadha

Srāvaṇyādi ghṛta, Balā ghṛta, Pāruṣaka ghṛta, Jivaniya ghṛta, Sthirādya ghṛta.

Taila

Sthirādya taila, Madhuparṇyādi taila, Sukumāraka taila, Amṛtādya taila, Mahāpadmaka taila, Khuḍḍāka-padmaka taila, Madhuka taila, Śatapāka-madhuka taila, Sahasrapāka and *Śatapāka balā taila, Piṇḍa taila, Vardhamāna pippali yoga.*

Anna

(C.Si.Ci. 29/50-54)

One should consume cereals like old Barley, Wheat, *Nivāra* (wild rice), *Śāli* and *Ṣaṣṭika* types of rice, soup of the meat of *Viṣkira* (gallinaceous) and *Pratuda* (pecker) birds; soup of *Āḍhaki, Caṇaka, Mudga, Masūra* and *Makuṣṭha* added with ghee in liberal quantity; leafy vegetable like *Suniṣaṇṇaka*, tender branches of *Vetra, Kākamāci, Śatāvari, Vāstuka, Upodikā* and *Sauvarcalā* fried with ghee. Meat Soup, Milk of Cow, Buffalo & Goat.

Vihāra

Warm poultices, pouring liquids on the body, warm application of paste of drug, residing in chamber devoid of heavy breeze, which is large & pleasing to the mind, use of soft pillows and bed & mild massaging.

The pigeon peas, grams, *Mudga*, lentil and *Samakuṣṭhakā* cooked along with *Ghṛtam* are considered to form good diet in cases of *Vāta Rakta*. Also eating old barley, wheat, *Nivāra* (*Tṛṇadhānya*), *Śāli* rice, *Ṣaṣṭhi* rice and milk of cow, buffalo and goats are also useful for the patients of the disease. (B.R. 27/5-6)

One should get the ointment, *Abhyañjana*, *Pariṣeka* and *Pralepa* therapies in cases of *Uttāna Vāta Rakta* (initial stage of gout). When the disease is serious, one should take treatment by *Sneha Pāna* (taking oily eatables), *Āsthāpana Basti* therapy and *Virecana* (purgation) therapy. One can process the blood letting therapy using needle, leeches, sharp horn or sharp part of the plants. Generally, ointment of *Śatadhauta Ghṛtam* and bathing with milk of sheep is useful in such cases. Massaging through oil medicated by muskmelon, white sandalwood, *Śinśapa* wood, *Aguru*, *Devadāru* and *Saral* wood is also helpful. Other useful edibles in this case include barley, *Ṣaṣṭhi*, *Nīvāra*, *Kalamā* and *Aruṇaśāli* varieties of rice, wheat, pulses of gram, kidney beans, pigeon peas and *Makuṣṭha*, milks of goat, buffalo and cow and non-vegetable preparations prepared out of the meats of peacock, hen, duck, parrots, pigeon, quails and the like; also good are the leafy vegetables involving *Upodikā*, *Kākamācī*, *Vetāgra*, pumpkin, *Vāstuka*, *Kāravellaka* and the like. (B.R. 27/195-202)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Avoid sexual intercourse, anger and food which are hot in potency, sour & salty, day sleep, food which brings more moisture inside & difficult for digestion. All etiological factors.

A patient of *Vāta-Rakta* should avoid the following: sleeping during daytime, exercising, indulging in intercourse, eating materials which are pungent, warm, *Gurupāki*, *Abhiṣyandi* (yogurt etc.), salty and bitter. (B.R. 27/7)

A patient of *Vāta-Rakta* should avoid the following: sleeping during daytime, enjoying heat of fire, exercising or wrestling, staying under sun, indulging in sexual activities, *Urdi*, *Kulattha*, *Niśpāva* (*Sema*) peas, *Kṣāra* products, flesh of aquatic and marshy animals (from the *Anūpa Deśa*), taking incompatible eatables, yogurt, sugarcane, liquor, *Piṇyāka*, cake of sesame seeds, sour and bitter drinks, *Kāñji*, items that are pungent, hot, heavy to digest and carminative (*Abhiṣyandi*). (B.R. 27/203-205).

Differential diagnosis of āmavāta

Sl. No.	***Points***	***Āmavāta***	***Sandhigata vāta***	***Vātarakta***	***Ūrusthambha***	***Sandhika sannipāta jvara***	***Kroṣṭukṣira***
1.	*Nidān* (Hetu)	*Āma & vāta kara āhara vihāra*	*Vātakara āhara vihāra*	*Vāta & rakta kara āhara vihāra*	*Kapha kara & Vātakar*	*Tṛdoṣa kara*	*Vāta & rakta kara vihāra*
2.	Dosic involvement	*Tṛdoṣa* mainly *vāta & kapha*	Mainly *vātadoṣa*	*Vāta & rakta*	*Kapha vāta*	*Tṛdoṣa*	*Vāta & rakta*
3.	*Pradhān* factors	*Āma* plays vital role with *vāta*	*Vāta* plays dominant role	*Vāta & rakta* both takes active part	*Kapha* dominates	*Tṛdoṣa*	*Vāta* dominates
4.	Involvement of *sandhi*	Intiates from big joints	starts from big joints	starts from small joints	*Uruśakti*	all *sandhi*	*Jānu sandhi*
5.	*Śūla* (Nature of Pain)	Nature of pain is like that of scorpion sting	*Bheda, Todana vāta pida*	*Bheda, Todana śpuranvā pida (Akhorviṣa)*	-	-	*Toda Bheda Mahārūja*
6.	*Rogādhikār* separate	Described as a *vāta vyādhi* disease entity	Included under disease	Separate disease	Separate disease entity	Included under *jvara roga*	Included under *vāta vyādhi*
7.	*Pratyātmā lakṣaṇa*	*Koṣṭha, Trika, Sandhiśūla* and stabdata	*Sandhi stadata śūla atopa*	Pain starts from *pada mūla*	Main site is the Urusakti	*Sandhisotha, Jvara, Nidrānās kāsa, stimita*	*Jānusotha* as like *Kroṣṭukṣira* (jacklehead) *Mahārūja*
8.	*Śūla Prakṛtī*	Radiating pain	oftenly radiates	Usually radiating pain is not *noticed*	Confined in the *ūru* only	Burning pain	Restricted to *Jānu sandhi* only
9.	*Upaśaya & Unpaśaya*	*Guggulu* preparation gives maximum benefit Intially snehana is contra indicated	Gives benefit snehana is the best therapy	It will not give much benefit Snehana Indicated	Gives benefit Snehana contra indicated	-	Give benefit
10.	*Rogamārga*	*Marmasthi sandhi* mainly *kapha sthānas*	*Sandhis*	*Marmasthi sandhi & sakhas*	*Uru* only	-	*Jānu* only

MCQs

1. *Adhya-roga* according to *Vagbhata* is
   a. *Ama-Vata*
   b. *Uru-Sthambha*
   c. *Vata-Rakta*
   d. *Meda-Kaphaavrutavata*
2. वर्जयेच्चैव सङ्कोचकरमिन्द्रियतापनम् । *are the features indicating asadhya lakshana of*
   a. *Ama-Vata*
   b. *Uru-Sthambha*
   c. *Vata-Rakta*
   d. *Meda-Kaphaavrutavata*
3. Which disease is having premonitory signs similar to *Kushta*
   a. *Krimi* b. *Vatarakta*
   c. *Visarpa* d. *Vidradhi*
4. *Bhutvabhutvapranasyatimuhuravirbhavanthi* is
   a. *Rupa of Vatarakta*
   b. *Purvarupa of Vatarakta*
   c. *Samanyalakshana of Vatarakta*
   d. *Rupa of Urusthambha*
5. *GambheeraVataraktafeatures* include all except
   a. *Swayadhu*
   b. *Twaktamra*
   c. *Karotikhanjampangum*
   d. *Both b & c*
6. *Chimichimayana* is the feature of which *doshika Vatarakta*
   a. *Vata* b. *Pitta*
   c. *Raktha* d. *Kapaha*

Answer

1.	c	2.	b	3.	b
4.	b	5.	b	6.	c

CHAPTER 47

Immunological & Metabolic Disorders

Rheumatic fever

- Rheumatic fever is an inflammatory disease that can involve the heart, joints, skin, and brain. It can develop as a complication of inadequately treated streptococcal infection of throat or scarlet fever. Streptococcal infection of throat and scarlet fever are caused by an infection with streptococcus bacteria.
- Rheumatic fever occurs in about 325,000 children each year and about 33.4 million people currently have rheumatic heart disease. Those who develop RF are most often between the ages of 5 and 14, with 20% of first-time attacks occurring in adults.
- The disease is most common in the developing world and among indigenous peoples in the developed world.
- Rheumatic fever can cause permanent damage to the heart, including damaged heart valves and heart failure. Treatments can reduce damage from inflammation, reduces pain and other symptoms and prevent the recurrence of complications

Causes

Rheumatic fever can occur after an infection of the throat with a bacterium called group A streptococcus. Group A streptococcus infections of the throat causes strep throat or, less commonly, scarlet fever. Group A streptococcus infections of the skin or other parts of the body rarely trigger rheumatic fever.

The link between strep infection and rheumatic fever isn't clear, but it appears that the bacterium trigger the immune system. The strep bacterium contains a protein similar to one found in certain tissues of the body. So, immune system cells that would normally target the bacterium may treat the body's own tissues. Particularly tissues of the heart, joints, skin and central nervous system are main target of such type of infection. This immune system reaction results in inflammation.

Pathogenesis of Rheumatic Fever

Group A β hemolytic streptococcal infection
↓
Pharyngitis
↓
Post pharyngeal infection (3 week)
↓
Production of antibody against streptococcal cell antigen
↓
Connective tissue of the heart, blood vessels, joints and subcutaneous tissues have antigen similarity to streptococcal cell antigen
↓
Antigen & antibody reactions
↓
Rheumatic fever with cardiac damage & other problems
like polyarthritis, chorea etc.

Clinical features

Rheumatic fever symptoms vary. Patients have few symptoms or several and symptoms can change during the course of the disease. The onset of rheumatic fever usually occurs about two to four weeks after a streptococcal throat infection. Followings are the common features of rheumatic fever.

- Fever
- Painful and tender joints - most often in the knees, ankles, elbows and wrists
- Pain in one joint that migrates to another joint
- Red, hot or swollen joints
- Small, painless bumps (nodules) beneath the skin
- Chest pain
- Heart murmur
- Fatigue
- Flat or slightly raised, painless rash with a

ragged edge (erythema marginatum)

- Jerky, uncontrollable body movements (Sydenham chorea, or St. Vitus' dance) - most often in the hands, feet and face
- Outbursts of unusual behavior, such as crying or inappropriate laughing, that accompanies Sydenham chorea

Risk factors

Factors that can increase the risk of rheumatic fever include:

- **Family history.** A gene or genes that might make them more likely to develop rheumatic fever.
- **Type of strep bacteria.** Certain strains of streptococcal bacteria are more likely to contribute to rheumatic fever than other strains.
- **Environmental factors.** A greater risk of rheumatic fever is associated with overcrowding, poor sanitation and other conditions.

Complications

Rheumatic fever can cause long-term complications in certain situations. One of the most prevalent complications is rheumatic heart disease. Other heart conditions include:

- Valve stenosis, which is a narrowing of a valve.
- Valve regurgitation, which is a leak in the valve that causes blood to flow in the wrong direction.
- Heart muscle damage, which is an inflammation that can weaken the heart muscle and decrease the heart's ability to pump blood effectively.
- Atrial fibrillation, which is an irregular heart beat in the upper chambers of the heart.
- Heart failure, which happens when the heart can no longer pump blood to all parts of the body.

Diagnostic criteria

Modified Jones criteria were first published in 1944 by T. Duckett Jones, MD. According to revised Jones criteria, the diagnosis of rheumatic fever can be made when two of the major criteria, or one major criterion plus two minor criteria, are present along with evidence of streptococcal infection: elevated or rising antistreptolysin O titre or DNAase. Signs of a preceding streptococcal infection include: recent scarlet fever, raised antistreptolysin O or other streptococcal antibody titre, or positive throat culture.

Major criteria

- **Polyarthritis:** A temporary migrating inflammation of the large joints, usually starting in the legs and migrating upwards.
- **Carditis:** Inflammation of the heart muscle (myocarditis) which can manifest as congestive heart failure with shortness of breath, pericarditis with a rub, or a new heart murmur.
- **Subcutaneous nodules:** Painless, firm collections of collagen fibers over bones or tendons. They commonly appear on the back of the wrist, the outside elbow, and the front of the knees.
- **Erythema marginatum:** A long-lasting reddish rash that begins on the trunk or arms as macules, which spread outward and clear in the middle to form rings, which continue to spread and coalesce with other rings, ultimately taking on a snake-like appearance. This rash typically spares the face and is made worse with heat.
- **Sydenham's chorea (St. Vitus' dance):** A characteristic series of involuntary rapid movements of the face and arms. This can occur very late in the disease for at least three months from onset of infection.

Minor criteria

- Fever of 38.2–38.9 ^{0}C (100.8–102.0^{0}F)
- Arthralgia: Joint pain without swelling (it cannot be included if polyarthritis is present as a major symptom)
- Raised erythrocyte sedimentation rate or C reactive protein
- Leukocytosis
- ECG showing features of heart block, such as a prolonged PR interval (Cannot be included if carditis is present as a major symptom)

- Previous episode of rheumatic fever or inactive heart disease

Rheumatoid arthritis (RA)

- Rheumatoid arthritis (RA) is the most common persistent inflammatory arthritis, occurring throughout the world and in all ethnic groups.
- The prevalence is lowest in black Africans and Chinese, and highest in Pima Indians. In Caucasians, it is 1.0-1.5%, with a female to male ratio of 3:7.
- The clinical course is prolonged, with intermittent exacerbations and remissions.
- Factors that associate with a poorer prognosis are disability at presentation, female gender, involvement of MTP joints, smoking and a positive RF and anti-CCP.
- It is hoped that the prognosis of RA will improve as more aggressive early treatment is used but this has yet to be demonstrated.

Etiology

- Both genetic and environmental factors appear to be involved in the pathogenesis of RA.
- The concordance rate of RA is higher in monozygotic (12-15%) than in dizygotic twins (3%), and there is an increased frequency of disease in first-degree relatives of patients.

3. Up to 50% of the genetic susceptibility is due to genes in the HLA region.
4. HLA-DR4 is the major susceptibility haplotype in most ethnic groups, occurring in 50-75% of Caucasian patients with RA compared to 20-25% of the normal population.
5. Although it is thought that RA may be triggered by an infectious agent in a genetically susceptible host, a specific pathogen has not been identified.
6. Susceptibility is increased post-partum and by breastfeeding.
7. Cigarette smoking is a strong risk factor for developing RA and also associates with greater severity.

Pathophysiology

Causative factors

↓

Leading to infiltration of the synovial membrane with lymphocytes, plasma cells and macrophages

↓

CD4+ T cells play a central role by interacting with other cells in the synovium.

↓

Activated T cells stimulate B cells to produce immunoglobulins including RF, and macrophages to produce proinflammatory cytokines.

↓

These act on endothelium, synovial fibroblasts, bone cells and chondrocytes

↓

Causes swelling and congestion of the synovial membrane and destruction of bone, cartilage and soft tissues

↓

Inflammatory granulation tissue (pannus) spreads over and under the articular cartilage, which is progressively eroded and destroyed

↓

Rheumatoid arthritis

↓

Later, fibrous or bony ankylosis may occur.

↓

Muscles adjacent to inflamed joints atrophy and may be infiltrated with lymphocytes.

↓

Rheumatoid nodules consist of a central area of fibrinoid material surrounded by a palisade of proliferating mononuclear cells.

↓

Similar granulomatous lesions may occur in the pleura, lung, pericardium and sclera.

↓

Immunofluorescence confirms RF synthesis by plasma cells in synovium and lymph nodes.

Clinical features

1. It is characterised by pain, joint swelling and stiffness affecting the small joints of the hands, feet and wrists.

2. Large joint involvement, systemic symptoms and extra-articular features may also occur.

Criteria for diagnosis of rheumatoid arthritis-American rheumatism Association 1988 revision

Diagnosis of RA is made with four or more of the following :

1. Morning stiffness (> 1 hr)
2. Arthritis of three or more joint areas
3. Arthritis of hand joints
4. Symmetrical arthritis
5. Rheumatoid nodules
6. Rheumatoid factor
7. Radiological changes

By convention, symptoms need to have persisted for more than or equal to 6 weeks for the diagnosis of RA.

The 2010 ACR/EULAR classification criteria for RA

According to the ACR/EULAR criteria, patients who should be tested are those (1) who have at least 1 joint with definite clinical synovitis and (2) whose synovitis is not better explained by another disease (lupus, psoriatic arthritis, or gout).

This is a score-based algorithm for RA that incorporates the following 4 factors :

- Joint involvement
- Serology test results
- Acute-phase reactant test results
- Patient self-reporting of the duration of signs and symptoms

The maximum number of points possible is 10. A classification of definitive RA requires a score of 6/10 or higher. Patients with a score lower than 6/10 should be reassessed over time.

A. **Joint involvement consists of swelling or tenderness upon examination :**

- 1 large joint (ie, shoulders, elbows, hips, knees, ankles) = 0 points
- 2-10 large joints = 1 point
- 1-3 small joints (with or without involvement of large joints), such as MCP, PIP, second to fifth MTP, thumb interphalangeal (IP), and wrist joints = 2 points
- 4-10 small joints (with or without involvement of large joints) = 3 points
- More than 10 joints (at least 1 small joint, plus any combination of large and additional small joints or joints such as the temporomandibular, acromio-clavicular, or sternoclavicular) = 5 points

B. At least 1 serology test result is needed for RA classification

Points are allocated as follows:

- Negative rheumatoid factor (RF) and negative anti citrullinated protein antibody (ACPA) tested as anti cyclic citrullinated peptide [anti-CCP]) = 0 points
- Low-positive RF or low-positive ACPA = 2 points
- High-positive RF or high-positive ACPA = 3 points

C. At least 1 test acute-phase reactant test result is needed for classification.

- Normal C-reactive protein (CRP) and normal erythrocyte sedimentation rate (ESR) = 0 points
- Abnormal CRP or abnormal ESR = 1 point

D. Points for the patient's self-reporting of the duration of signs or symptoms of synovitis in clinically involved joints are allocated as follows:

- Shorter than 6 weeks = 0 points
- 6 weeks or longer = 1 point

Assessment of disease activity in rheumatoid arthritis

1. Clinical

- Pain
- Visual analogue scale
- Early morning stiffness
- Minutes
- Joint tenderness
- Number of inflammed joints

2. Laboratory

- Acute phase proteins
- Erythrocyte sedimentation rate

- Plasma viscosity
- Presence of anti-CCP antibodies

3. **Articular index**
    - Plain radiography of joints
    - USG & MRI of joints

Grading of function in rheumatoid arthritis

I	Fit for all activities	No handicap
II	Moderate restriction	Independent despite handicap and limitation of joint movement
III	Marked restriction	Limited self-care. Some assistance required
IV	Confined to chair or bedbound	Largely incapacitated and dependent

Physical Examination

It is important to assess the following signs :

- Stiffness
- Tenderness
- Pain on motion
- Swelling
- Deformity
- Limitation of motion
- Extra-articular manifestations
- Rheumatoid nodules

Hands

1. 'Swan neck' deformity
2. The boutonniere or 'button hole' deformity
3. Z deformity of the thumb
4. Dorsal subluxation of the ulna at the distal radio-ulnar joint is common

Foot

1. Dorsal subluxation of the MTP joints may result in 'cock-up' toe deformities.
2. In the hindfoot, calcaneovalgus (eversion) is the most common deformity, reflecting damage to the ankle and subtalar joint.
3. Popliteal (Baker's) cysts usually occur in combination with knee synovitis.
4. Rupture, often induced by knee flexion in the presence of a large effusion, leads to calf pain and swelling.

Investigations

1. ESR and CRP are usually raised (acute phase response)
2. Rheumatoid Factor and anti-CCP antibodies
3. Plain X-rays of the hands, wrist and feet are useful
4. Peri articular osteoporosis is common during the early stage of disease
5. Non-proliferative marginal joint erosions on X-ray are characteristic but uncommon within the first year.
6. Ultrasound and MRI are more sensitive than X-rays at detecting early erosions.
7. Patients who are suspected of having atlanto-axial disease should have lateral X-rays taken in flexion and extension, and the degree of cord compression should be established with MRI.
8. In patients with Baker's cyst, Doppler ultrasound and an arteriogram may be required to establish the diagnosis, since deep Venous thrombosis (DVT) and Baker's cyst may coexist.

Outcome measures in rheumatoid arthritis

- Funtional
- Grip strength
- Activities of daily living
- Functional index
- Health Status Questionnaire

Dimensions	Subdimensions	Components (e.g.)
Death	Upper lower	Grip, feed, walk, climb
Disability	Physical	Pain, fatigue,
Discomfort	Psychological	Depression, anxiety
Iatrogenic	Medical	Dyspepsia, haemorrhage
	Surgical	Prosthesis infection and loosening
Economic	Direct	Drugs, consultations,
	Indirect	Loss of work, social

Systemic Lupus Erythematosus (SLE)

- Systemic lupus erythematosus (SLE) is an autoimmune disorder characterized by antibodies to nuclear and cytoplasmic antigens, multisystem inflammation, protean clinical manifestations, and a relapsing and remitting course.
- More than 90% of cases of SLE occur in women, frequently starting at childbearing age.

Pathophysiology and Etiology

- SLE is an autoimmune disorder characterized by multisystem inflammation with the generation of autoantibodies.
- Although the specific cause of SLE is unknown
- Multiple factors are associated with the development of the disease, including genetic, epigenetic, ethnic, immunoregulatory, hormonal, and environmental factors.

Clinical presentation-Signs and symptoms of SLE

- SLE is a chronic inflammatory disease that can affect almost any organ system, although it mainly involves the skin, joints, kidneys, blood cells, and nervous system.
- Its presentation and course are highly variable, ranging from indolent to fulminant.

In childhood-onset SLE, the following clinical manifestations are more commonly found than in adults :

- Malar rash
- Ulcers/mucocutaneous involvement
- Kidney involvement, proteinuria, urinary cellular casts
- Seizures
- Thrombocytopenia
- Hemolytic anemia
- Fever
- Lymphadenopathy.

In adults, Raynaud pleuritis and sicca are twice as common as in children and adolescents.

- The classic presentation of a triad of fever, joint pain, and rash in a woman of childbearing age should prompt investigation into the diagnosis of SLE.

Patients may present with any of the following manifestations:

- Constitutional (eg, fatigue, fever, arthralgia, weight changes)
- Musculoskeletal (eg, arthralgia, arthropathy, myalgia, frank arthritis, avascular necrosis)
- Dermatologic (eg, malar rash, photosensitivity, discoid lupus)
- Renal (eg, acute or chronic kidney failure, acute nephritic disease)
- Neuropsychiatric (eg, seizure, psychosis)
- Pulmonary (eg, pleurisy, pleural effusion, pneumonitis, pulmonary hypertension, interstitial lung disease)
- Gastrointestinal (eg, nausea, dyspepsia, abdominal pain)
- Cardiac (eg, pericarditis, myocarditis)
- Hematologic (eg, cytopenias such as leukopenia, lymphopenia, anemia, or thrombocytopenia)

Note- In patients with suggestive clinical findings, a family history of autoimmune disease should raise further suspicion of SLE.

Ancillary investigations & Diagnosis of SLE

- The diagnosis of SLE is based on a combination of clinical findings and laboratory evidence.
- Familiarity with the diagnostic criteria helps clinicians to recognize SLE and to subclassify this complex disease based on the pattern of target-organ manifestations.
- The American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) published new criteria for the classification of SLE in 2019. These criteria represent current concepts of SLE, and have excellent specificity and sensitivity.

The American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR)

- The EULAR/ACR classification requires an antinuclear antibody (ANA) titer of at least 1:80 on HEp-2 cells or an equivalent positive test at least once; otherwise, the patient is considered not to have SLE.

- If it is present, 22 "additive weighted" classification criteria are considered, comprising seven clinical domains and three immunologic domains.
- Each criterion is assigned points, ranging from 2 to 10. Patients with at least one clinical criterion and 10 or more points are classified as having SLE.

EULAR/ACR Clinical Domains and Criteria for SLE

Domain	Criteria	Points
Constitutional	Fever	2
Hematologic	Leukopenia Thrombocytopenia Autoimmune hemolysis	3 4 4
Neuropsychiatric	Delirium Psychosis Seizure	2 3 5
Mucocutaneous	Non-scarring alopecia Oral ulcers Subacute cutaneous or discoid lupus Acute cutaneous lupus	2 2 4 6
Serosal	Pleural or pericardial effusion Acute pericarditis	5 6
Musculoskeletal	Joint involvement	6
Renal	Proteinuria > 0.5 g/24 h Kidney biopsy class II or V lupus nephritis Kidney biopsy class III or IV lupus nephritis	4 8 10

Note the following :

- A criterion should not be counted if there is a more likely explanation for it than SLE
- Occurrence of a criterion on at least one occasion is sufficient
- Criteria need not occur simultaneously
- Within each domain, only the highest-weighted criterion is counted toward the total score

Testing

Laboratory studies used in the diagnosis of SLE are as follows:

- CBC with differential
- Serum creatinine
- Urinalysis with microscopy
- ESR or CRP level
- Complement levels
- Liver function tests
- Creatine kinase assay
- Spot protein/spot creatinine ratio
- Autoantibody tests

Autoantibody tests

Autoantibody Tests for SLE

Test	Description
ANA	Screening test; sensitivity 95%; not diagnostic without clinical features
Anti-dsDNA	High specificity; sensitivity only 70%; level is variable based on disease activity
Anti-Sm	Most specific antibody for SLE; only 30-40% sensitivity
Anti-SSA (Ro)	Present in 15% of patients with
or Anti-SSB (La)	SLE and other connective-tissue diseases such as Sjogren syndrome; associated with neonatal lupus
Anti-ribosomal P	Uncommon antibodies that may correlate with risk for CNS disease, including increased hazards of psychosis in a large inception cohort, although the exact role in clinical diagnosis is debated.
Anti-RNP	Included with anti-Sm, SSA, and SSB in the ENA profile; may indicate mixed connective-tissue disease with overlap SLE, scleroderma, and myositis
Anticardiolipin	IgG/IgM variants measured with

	ELISA are among the antiphospholipid antibodies used to screen for antiphospholipid antibody syndrome and pertinent in SLE diagnosis
Lupus anticoagulant	Multiple tests (eg, direct Russell viper venom test) to screen for inhibitors in the clotting cascade in antiphospholipid antibody syndrome
Direct Coombs test	Coombs test–positive anemia to denote antibodies on RBCs
Anti-histone	Drug-induced lupus ANA antibodies are often of this type (eg, with procainamide or hydralazine; p-ANCA–positive in minocycline-induced drug-induced lupus)

ANA = antinuclear antibody; CNS = central nervous system; ds-DNA = double-stranded DNA; ELISA = enzyme-linked immunoassay; ENA = extractable nuclear antigen; Ig = immunoglobulin; p-ANCA = perinuclear antineutrophil cytoplasmic antibody; RBCs = red blood cells; RNP = ribonucleic protein; SLE = systemic lupus erythematosus; Sm = Smith; SSA = Sjogren syndrome A; SSB = Sjogren syndrome B.

Imaging studies

The following imaging studies may be used to evaluate patients with suspected SLE:

- Joint radiography
- Chest radiography and chest CT scanning
- Echocardiography
- Brain MRI/MRA
- Cardiac MRI

Procedures

Procedures that may be performed in patients with suspected SLE include the following:

- Arthrocentesis
- Lumbar puncture
- Kidney biopsy

Ankylosing spondylitis

1. Ankylosing spondylitis (AS) is characterised by a chronic inflammatory arthritis predominantly affecting the sacroiliac joints and spine, which can progress to bony fusion of the spine.
2. It has a peak onset in the second and third decades
3. Male female ratio of about 3:1.
4. In Europe, more than 90% of those affected are HLA-B27-positive.
5. The overall prevalence is less than 0.5% in most communities.
6. Over 75% of patients are able to remain in employment and enjoy a good quality of life.
7. Even if severe ankylosis develops, functional limitation may not be marked as long as the spine is fused in an erect posture

Etiopathogenesis

It result from exposure to a common environmental pathogen in genetically susceptible individuals

↓

Although no specific trigger has been identified

↓

Chronic prostatitis is more common than expected but appears to be non-infective in origin

↓

Increased faecal carriage of Klebsiella aerogenes occurs in patients with established AS

↓

May relate to exacerbation of both joint and eye disease.

Clinical features

1. The onset is usually insidious, with recurring episodes of low back pain and marked stiffness.
2. Radiation of pain to the buttocks or posterior thighs may be misdiagnosed as sciatica.
3. Unlike mechanical back pain, symptoms extend over many segments and are axial and symmetrical in distribution.
4. Symptoms are most marked in the early morning and after inactivity
5. Symptoms are relieved by movement.
6. Although the lumbosacral area is usually the first and worst affected region

7. Some patients present with mainly thoracic or neck symptoms.
8. The disease tends to ascend the spine slowly and eventually
9. After several years, the whole spine may be affected.
10. As the spine becomes progressively ankylosed, spinal rigidity and secondary osteoporosis predispose to spinal fracture.
11. Spinal cord compression is rare.

Physical examination findings initially

1. Failure to obliterate the lumbar lordosis on forward flexion
2. Restriction of movements of the lumbar spine in all directions
3. Possible pain on sacroiliac stressing

Physical examination findings of progressive disease

1. Stiffness increases throughout the spine
2. Chest expansion frequently becomes restricted.
3. Spinal fusion varies in its extent and in most cases does not cause a gross flexion deformity.
4. A few cases develop marked kyphosis of the dorsal and cervical spine that may interfere with forward vision.
5. This may prove incapacitating, especially when associated with fixed flexion contractures of hips or knees.
6. Pleuritic chest pain aggravated by breathing is common and results from costovertebral joint involvement.
7. Plantar fascitis, Achilles tendinitis and tenderness over bony prominences such as the iliac crest and greater trochanter are common, reflecting inflammation at the sites of tendon insertions (enteritis).

Peripheral arthritis

1. Up to 40% of patients have peripheral arthritis.
2. It is usually asymmetrical at first, mainly affecting hips, knees, ankles or shoulders.
3. Involvement of a peripheral joint, most commonly ankle, knee or elbow, may precede the development of spinal symptoms in around 10% of cases.
4. In a further 10%, symptoms begin in childhood as one variety of pauciarticular juvenile idiopathic arthritis.

Extra-articular disease

1. Fatigue is often a major complaint and may result from both chronic interruption of sleep due to pain, as well as chronic systemic inflammation.
2. Acute anterior uveitis is the most common extra-articular feature.
3. Conjunctivitis
4. Prostatitis : usually asymptomatic
5. Aortic incompetence
6. Mitral incompetence
7. Cardiac conduction defects
8. Pericarditis
9. Amyloidosis
10. Atypical upper lobe pulmonary fibrosis

Investigations

A. **Blood**

1. The ESR and CRP are usually raised in active disease.
2. RF and other autoantibodies are usually negative.
3. Testing for HLA-B27 can be a helpful investigation for pauciarticular juvenile idiopathic arthritis but is unhelpful in adults with spinal symptoms.

B. **X-ray**

1. X-ray changes are characteristic but may take year to develop.
2. Sacroiliitis is often the first abnormality beginning in the lower synovial parts of the joints with irregularity and loss of cortical margins, widening of the joint space and subsequently sclerosis, narrowing and fusion.
3. Lateral thoracolumbar spine X-rays may show anterior 'squaring' of vertebrae due to erosion and sclerosis of the anterior corners and periostitis of the waist.

4. Bridging syndesmophytes are fine and symmetrical and follow the outermost fibres of the annulus.
5. Ossification of the anterior longitudinal ligament and, facet joint fusion may also be visible and may result in the typical 'bamboo' spine
6. Erosive changes may be seen in the symphysis pubis, the ischial tuberosities and peripheral joints.
7. Osteoporosis and atlanto-axial dislocation can occur as late features.

C. MRI

MRI is more sensitive for detection of early sacroiliitis, but is seldom required.

Gout

1. Gout is a true crystal deposition disease, and is defined as the pathological reaction of the joint or periarticular tissues to the presence of monosodium urate monohydrate (MSU) crystals.
2. It results from an increased body pool of urate with hyperuricemia.
3. It typically is characterized by episodic acute and chronic arthritis caused by deposition of MSU crystals in joints and connective tissue tophi and the risk for deposition in kidney interstitium or uric acid nephrolithiasis.

Epidemiology

- The prevalence of gout varies between populations but is approximately 1-2%, with a strong male predominance (> 5:1).
- It is the most common inflammatory arthritis in men and in older women.
- Prevalence increases with increasing serum uric acid (SUA) and with age.
- The incidence and prevalence of gout have increased in many countries in recent decades, in parallel with increased longevity and the higher prevalence of metabolic syndrome, of which hyperuricaemia is an integral component.

Hyperuricaemia

- It is defined as an SUA level greater than 2 standard deviations above the mean for the population.
- Only a minority of hyperuricaemic people develop gout, emphasising the importance of tissue factors in crystal formation.

Aetiology Primary gout

- About one-third of the body uric acid pool is derived from dietary sources and two-thirds from endogenous purine metabolism
- The concentration of uric acid in body fluids depends on the balance between its synthesis and elimination by the kidneys (two-thirds) and gut (one-third).
- Purine nucleotide synthesis and degradation are regulated by a network of enzyme pathways; xanthine oxidase catalyses the end conversion of hypoxanthine to xanthine and then xanthine to uric acid.
- In over 90% of patients with primary gout, hyperuricaemia results from an inherited defect in fractional uric acid excretion which impairs their ability to increase urate excretion in response to a purine load ('underexcretors').

Risk factors and associations for primary gout include

- Metabolic syndrome, high alcohol intake (predominantly beer which contains guanosine), and diets relatively high in red meat or fructose or relatively low in vitamin C or coffee.

Secondary gout

- Secondary gout results from hyperuricaemia due to renal impairment or chronic diuretic use.

Factors that predispose to chronic hyperuricaemia and gout

Diminished renal excretion (common)

- Inherited isolated renal tubular defect ('under-excretors')
- Renal failure
- Chronic drug therapy

  Thiazide and loop diuretics
  Low-dose aspirin Ciclosporin
  Pyrazinamide

- Lead toxicity (e.g. in 'moonshine' drinkers)
- Lactic acidosis (alcohol)

Increased production of uric acid (uncommon)

- Increased purine turnover

  Chronic myeloproliferative or lymphoproliferative disorders (e.g. polycythaemia, chronic lymphatic leukaemia)
- Increased de novo synthesis ('over-producers')
- Unidentified abnormality (most common)
- Specific enzyme defect (rare)
- Hypoxanthine-guanine phosphoribosyl transferase deficiency
- Phosphoribosyl pyrophosphate synthetase over-activity
- Glucose-6-phosphatase deficiency

Acute and Chronic Arthritis

1. Acute arthritis is the most common early clinical manifestation of gout
2. Usually, only one joint is affected initially, but polyarticular acute gout can occur in subsequent episodes.
3. Joints involvement
   - The first MTP joint is affected in over 50% of cases'podagra' ('seizing the foot').
   - Other common sites are, in order of decreasing frequency, the ankle, midfoot, knee, small joints of hands, wrist and elbow.
   - The axial skeleton and large proximal joints are rarely involved and never as the first site.
   - **Typical attacks have the following characteristics :**
   - Extremely rapid onset, reaching maximum severity in just 2-6 hours, often waking the patient in the early morning severe pain, often described as the 'Worst pain ever
   - Extreme tenderness: the patient is unable to wear a socks or to let bedding rest on the joint
   - marked swelling with overlying red, shiny skin
   - Self-limiting over 5-14 days, with complete return to normality.
4. First episode
   - The first episode of acute gouty arthritis frequently begins at night with dramatic joint pain and swelling.
   - Joints rapidly become warm, red, and tender, with a clinical appearance that often mimics that of cellulitis.
   - Early attacks tend to subside spontaneously within 3–10 days, and most patients have intervals of varying length with no residual symptoms until the next episode.
5. Precipitating factors

   Dietary excess, trauma, surgery, excessive ethanol ingestion, hypouricemic therapy, and serious medical illnesses such as myocardial infarction and stroke
6. Heberden's or Bouchard's nodes
   - Inflamed Heberden's or Bouchard's nodes may be a first manifestation of gouty arthritis
7. Chronic nonsymmetric synovitis
   - After many acute mono- or oligoarticular attacks, a proportion of gouty patients may present with a chronic nonsymmetric synovitis, causing potential confusion with rheumatoid arthritis
   - Less commonly, chronic gouty arthritis will be the only manifestation, and, more rarely, the disease will manifest only as periarticular tophaceous deposits in the absence of synovitis.
   - Women represent only 5–20% of all patients with gout.
   - Premenopausal gout is rare; it is seen mostly in individuals with a strong family history of gout.

Laboratory Diagnosis

1. Definitive diagnosis requires identification of MSU crystals in the aspirate from a joint, bursa or tophus
2. During acute gouty attacks, needle-shaped MSU crystals typically are seen both intracellularly and extracellularly.
3. Synovial fluid leukocyte counts are elevated from 2000 to 60,000/L.
4. Effusions appear cloudy due to the increased numbers of leukocytes.
5. Large amounts of crystals occasionally produce a thick pasty or chalky joint fluid.

6. Arthrocentesis of these joints is a useful technique to establish the diagnosis of gout between attacks.
7. Serum uric acid levels can be normal or low at the time of an acute attack, as inflammatory cytokines can be uricosuric and effective initiation of hypouricemic therapy can precipitate attacks.
8. A 24-h urine collection for uric acid can, in some cases, be useful in assessing the risk of stones, elucidating overproduction or underexcretion of uric acid, and deciding whether it may be appropriate to use a uricosuric therapy.
   - Excretion of >800 mg of uric acid per 24 h on a regular diet suggests that causes of overproduction of purine should be considered.
9. Other tests
   - Urinalysis, serum creatinine, hemoglobin, white blood cell (WBC) count, liver function tests, and serum lipids should be obtained because of possible pathologic sequelae of gout
10. Radiographic Features
   - Cystic changes, well-defined erosions with sclerotic margins (often with overhanging bony edges), and soft tissue masses are characteristic features of advanced chronic tophaceous gout .
   - Ultrasound, CT and MRI are being studied and are likely to become more sensitive for early changes.

MCQs

1. Serum antibodies to cyclic citrullinated peptides (anti-CCPs) are now recognized to be a valuable biomarker of diagnostic and prognostic significance of
   a. Gouty arthritis
   b. Reactive arthritis
   c. Rheumatoid arthritis (RA)
   d. Ankylosing spondylitis
2. During acute attacks of which disease needle-shaped MSU crystals typically are seen both intracellularly and extracellularly
   a. Gouty arthritis
   b. Reactive arthritis
   c. Psoriatic arthritis
   d. Ankylosing spondylitis
3. Antinuclear antibodies (ANA) are positive in >98% of patients during the course of disease and High-titer IgG antibodies to double-stranded DNA and antibodies to the Sm antigen are both specific for
   a. SLE and, therefore, favor the diagnosis in the presence of compatible clinical manifestations
   b. RA and, therefore, favor the diagnosis in the presence of compatible clinical manifestations
   c. Gouty arthritis and, therefore, favor the diagnosis in the presence of compatible clinical manifestations
   d. Reactive arthritis and, therefore, favor the diagnosis in the presence of compatible clinical manifestations
4. Inflamed Heberden's or Bouchard's nodes may be a first manifestation of which arthritis
   a. Gout b. Osteoarthritis
   c. Rheumatoid arthritis d. Rheumatic fever
5. Foot deformities (such as hammertoes or clawtoes and Charcot foot) are seen in
   a. Rheumatic fever
   b. Rheumatoid arthritis
   c. Diabetes mellitus (DM)
   d. Ankylosing spondylitis

Answer

1.	c	2.	a	3.	a
4.	a	5.	b		

CHAPTER
48

Klaibya & Vandhyatva

क्लैब्य (Klaibya)

Varieties of Impotency

रेतोदोषोद्भवं क्लैब्यं यस्माच्छुद्ध्यैव सिध्यति ॥
ततो वक्ष्यामि ते सम्यगग्निवेश! यथातथम् ।
बीजध्वजोपघाताभ्यां जरया शुक्रसंक्षयात् ॥
क्लैब्यं संपद्यते । *C.S.Ci 30/153-155*

Since impotency is caused by the seminal morbidities, and it gets corrected by the purification of the semen, now, O Agniveśa ! I Shall appropriately describe [the etiology, signs and treatment of] this disease systematically. Impotency is of four types depending upon its causative factors as follows :

1. *Bījopaghātaja klaibya* (impotency caused by seminal diminution)
2. *Dhvajabhaṅgaja klaibya* (impotency caused by nonerectile phallus)
3. *Jarāja klaibya* (impotency caused by old age)
4. *Śukra-kṣayaja klaibya* (impotency caused by excessive loss of semen, i.e. by sexual intercourse).

सामान्य लक्षण (Sāmānya Lakṣaṇa)

Signs and Symptoms of Impotency in General

सङ्कल्पप्रवणो नित्यं प्रियां वश्यामपि स्त्रियम् ॥
न याति लिङ्गशैथिल्यात् कदाचिद्याति वा यदि ।
श्वासार्तः स्विन्नगात्रश्च मोघसङ्कल्पचेष्टितः ॥
म्लानशिश्नश्च निर्बीजः स्यादेतत् क्लैब्यलक्षणम् ।
सामान्यलक्षणं ह्येतद्विस्तरेण प्रवक्ष्यते ॥
C.S.Ci 30/155-157

Eventhough a man is constantly desirous of sexual intercourse with the partner who is co-operative, he, because of the looseness (absence of erection) of the phallus becomes incapable of performing the sexual act. Even if he rarely attempts sexual act, he gets afflicted with dyspnoea as well as perspiration in the body, and gets frustrated in his determined efforts. His phallus becomes loose (because of the lack of erection), and he does not ejaculate any semen. These are the general signs and symptoms of impotency. Specific signs and symptoms of impotency will, hereafter be, described in detail.

बीजोपघातज क्लैब्य लक्षण (Bījopaghātaja Klaibya Lakṣaṇa)

Etiology and Signs of Bījopaghātaja Klaibya

शीतरूक्षाल्पसंक्लिष्टविरुद्धाजीर्णभोजनात् ।
शोकचिन्ताभयत्रासात् स्त्रीणां चात्यर्थसेवनात् ॥
अभिचारादविस्रम्भाद्रसादीनां च संक्षयात् ।
वातादीनां च वैषम्यात्तथैवानशनाच्छ्रमात् ॥
नारीणामरसज्ञत्वात् पञ्चकर्मापचारतः ।
बीजोपघाताद्भवति पाण्डुवर्णः सुदुर्बलः ॥
अल्पप्राणोऽल्पहर्षश्च प्रमदासु भवेन्नरः ।
हृत्पाण्डुरोगतमककामलाश्रमपीडितः ॥
छर्द्यतीसारशूलार्तः कासज्वरनिपीडितः ।
बीजोपघातजं क्लैब्यं.....................................
C.S.Ci 30/158-162

In *Bījopaghātaja* type of *Klaibya* (impotency caused by the diminution of semen), the semen gets vitiated and diminished in quantity because of the following:

Intake of cold, ununctuous, scanty, polluted and mutually conradictory ingredients of food; intake of food before the previous meal is digested; giref, anxiety, fear and terror; excessive indulgence in sex with woman; *Abhicāra* (affliction by black magic); *Avisrambha* (suspicious nature); diminution of *Rasa* and other tissue elements; disharmony among *Vāta* and other *Doṣas;* fasting and fatigue; disliking for women; and improper administration of *Pañca-karma* (five elemination therapies).

Because of the seminal destruction (diminution) as a result of the aforesaid factors, the patient becomes pale in colour, very weak and low in vitality. He gets low excitement while meeting female parteners. He suffers from heart diseases, anemia, asthma, jaundice, physical exhaustion, vomiting, diarrhoea, colic pain, cough and fever.

ध्वजभङ्गज क्लैब्य हेतु (Dhvajabhaṅgaja Klaibya Hetu)

Etiology of Dhvajabhaṅgaja Klaibya

अत्यम्ललवणक्षारविरुद्धासात्म्यभोजनात् ।
अत्यम्बुपानाद्विषमात् पिष्टान्नगुरुभोजनात् ॥
दधिक्षीरानूपमांससेवनाद्व्याधिकर्षणात् ।
कन्यानां चैव गमनादयोनिगमनादपि ॥
दीर्घरोगां चिरोत्सृष्टां तथैव च रजस्वलाम् ।
दुर्गन्धां दुष्टयोनिं च तथैव च परिस्नुताम् ॥
ईदृशीं प्रमदां मोहाद्यो गच्छेत् कामहर्षितः ।
चतुष्पदाभिगमनाच्छेफसश्चाभिघाततः ॥
अधावनाद्वा मेढ्रस्य शस्त्रदन्तनखक्षतात् ।
काष्ठप्रहारनिष्पेषाच्छूकानां चातिसेवनात् ॥
रेतसश्च प्रतीघाताद्ध्वजभङ्गः प्रवर्तते ।

C.S.Ci 30/162-167

Intake of excessively sour, saline, alkaline, mutually antagonistic and unwholesome ingredients of food; intake of water in excess; taking meals irregularly; intake of pastry and heavy food habitually; intake of yoghurt, milk and meat of animals inhabiting marshy land; emaciation because of diseases; cohabitation with young virgin girls; sexual intercourse in parts other than vagina; because of excitement and ignorance, sexual intercourse with a woman who is suffering from chronic diseases, in continuation who has shunned sexual relationship for a long time, who is in menstruation, and whose vagina is offensive in smell, afflicted with diseases and has profuse discharge; sexual intercourse with quadruped animals; trauma to the phallus; not cleaning the phallus properly; injury to the phallus by weapons, teeth, nails, beating by a stick or compression; excessive use of *Śūkās* (a type of insect which is applied for the elongation of the phallus); and suppression of the urge for seminal ejaculation during sexual intercourse.

ध्वजभङ्गज क्लैब्य लक्षण (Dhvajabhaṅgaja Klaibya Lakṣaṇa)

Symptoms of Dhvajabhaṅgaja Klaibya

श्वयथुर्वेदना मेढ्रे रागश्चैवोपलक्ष्यते ॥
स्फोटाश्च तीव्रा जायन्ते लिङ्गपाको भवत्यपि ।
मांसवृद्धिर्भवेच्चास्य व्रणाः क्षिप्रं भवन्त्यपि ॥
पुलाकोदकसङ्काशः स्रावः श्यावारुणप्रभः ।
वलयीकुरुते चापि कठिनश्च परिग्रहः ॥
ज्वरस्तृष्णा भ्रमो मूर्च्छा च्छर्दिश्चास्योपजायते ।
रक्तं कृष्णं स्रवेच्चापि नीलमाविललोहितम् ॥
अग्निनेव च दग्धस्य तीव्रो दाहः सवेदनः ।
बस्तौ वृषणयोर्वाऽपि सीवन्यां वङ्क्षणेषु च ॥
कदाचित्पिच्छिलो वाऽपि पाण्डुः स्रावश्च जायते ।
श्वयथुर्जायते मन्दः स्तिमितोऽल्पपरिस्रवः ॥
चिराच्च पाकं व्रजति शीघ्रं वाऽथ प्रमुच्यते ।
जायन्ते क्रिमयश्चापि क्लिद्यते पूतिगन्धि च ॥
विशीर्यते मणिश्चास्य मेढ्रं मुष्कावथापि च ।
ध्वजभङ्गकृतं क्लैब्यमित्येतत् समुदाहृतम् ॥
एतं पञ्चविधं केचिद्ध्वजभङ्गं प्रचक्षते ।

C.S.Ci 30/168-176

Swelling, pain and redness of the phallus; appearance of acute boils, inflammation in *Penis*; fleshy growth in the phallus and its quick ulceration; exudation which appears like rice-water *(Pulakodaka)* or which is brownish black or pink in colour; circular and hard induration below the glance penis; fever, morbid thirst, giddiness, fainting and vomiting; discharge of red, black, blue, turbid and red coloured liquid from the urethra; acute burning sensation as if burnt by fire, and pain in the region of urinary bladder, testicles, perineal suture and groins; discharge of slimy and pale yellow liquid at times;mild swelling, numbness and scanty discharge; it takes long time to suppurate and may get abated quickly; appearance of maggots in the phallus; sloughing and foul smell of the phallus; and dropping of the glance penis or of the whole penis or of the testicles.

Thus, the impotency caused by *Dhvajabhaṅga* (morbidity of the phallus) is explained.

According to some physicians, this type of impotency cuased by *Dhvajabhaṅga* is of five varieties.

The five varieties of *Dhvajabhaṅga* are as follows :

1. *Vātaja Dhvajabhaṅga*
2. *Pittaja Dhvajabhaṅga*
3. *Kaphaja Dhvajabhaṅga*
4. *Raktaja Dhvajabhaṅga*
5. *Sānnipātika Dhvajabhaṅga*

जराज क्लैब्य हेतु एवं लक्षण (Jarāja Klaibya Hetu evam Lakṣaṇa)

जघन्यमध्यप्रवरं वयस्त्रिविधमुच्यते।
अतिप्रवयसां शुक्रं प्रायशः क्षीयते नृणाम्॥
रसादीनां संक्षयाश्च तथैवावृष्यसेवनात्।
बलवीर्येन्द्रियाणां च क्रमेणैव परिक्षयात्॥
परिक्षयादायुषश्चाप्यनाहाराच्छ्रमात् क्लमात्।
जरासंभवजं क्लैब्यमित्यैर्हेतुभिर्नृणाम्॥
जायते तेन सोऽत्यर्थं क्षीणधातुः सदुर्बलः।
विवर्णो दुर्बलो दीनः क्षिप्रं व्याधिमथाश्नुते॥
एतज्जरासंभवं हि............................

C.S.Ci. 30/176-180

Age of a person is divided into three parts, viz., *Jaghanya* (childhood), *Madhya* (adulthood) and *Pravara* (old age). In the old age, generally the semen gets diminished.

Impotency takes place in the old age because of the following : Diminution of tissue elements like *Rasa* (Plasma), etc.; constant use of ingredients which are detrimental to the vitality of a person; gradual diminution of strength, energy, power of senses and span of life; inability to take nourishing food, and physical as well as mental fatigue.

Because of the aforesaid factors, the tissue elements of the old man becomes diminished and excessively weak; his complexion becomes perverted; he becomes physically and mentally weak; and he succumbs to diffrent types of disease quickly.

It is stated above that in the old age, generally the semen gets diminished. Use of the word 'generally' *(Prāyaśaḥ)* is significant inasmuch as at times, the semen does not get diminished even in old age if the person is *Śukra-Sāra* (having the excellence of semen) and if he habitually takes aphrodisiac drugs.

क्षयज क्लैब्य (Kṣayaja Klaibya)

........................चतुर्थं क्षयजं शृणु।
अतीव चिन्तनाच्चैव शोकात्क्रोधाद्भयात्तथा॥
ईर्ष्योत्कण्ठामेदोद्वेगान् सदा विशति यो नरः।
कृशो वा सेवते रूक्षमन्नपानं तथौषधम्॥
दुर्बलप्रकृतिश्चैव निराहारो भवेद्यदि।
असात्म्यभोजनाच्चापि हृदये यो व्यवस्थितः॥
रसः प्रधानधातुर्हि क्षीयेताशु ततो नृणाम्।
रक्तादयश्च क्षीयन्ते धातवस्तस्य देहिनः॥
शुक्रावसानास्तेभ्योऽपि शुक्रं धाम परं मतम्।
चेतसो वाऽतिहर्षेण व्यवायं सेवतेऽति यः॥
तस्याशु क्षीयते शुक्रं ततः प्राप्नोति संक्षयम्।
घोरं व्याधिमवाप्नोति मरणं वा स गच्छति॥
शुक्रं तस्माद्विशेषेण रक्ष्यमारोयमिच्छता।

C.S.Ci 30/181-187

Now hear about the fourth type of impotency which is caused by the diminution of semen *(Kṣayaja)*. The semen in a man gets diminished because of the following factors;

Constant exposure to worry, grief, anger, fear, envy, anxiety, intoxication and nervousness; intake of ununctuous food, drinks and drugs by an emaciated person; fasting by a person who is weak in nature; and intake of unwholesome food.

क्षयज क्लैब्य संप्राप्ति (Kṣayaja Klaibya Samprāpti)

By the aforesaid factors, *Rasa* (plasma) which is the primary tissue element and which is located in the heart, gets diminished soon. As a result of this, other tissue elements begining from *Rakta* (blood) upto semen get diminished in that person. Among all these tissue elements, semen *(Śukra)* which is the final product is the most important.

If a person because of excessive mental

excitement indulge in sexual intercourse in excess, his semen gets diminished soon, and he gets emaciated. He succumbs to serious disease, and even death. Therefore, a person desirous of good health should specially preserve his semen.

साध्यासाध्यता (Sādhyāsādhyatā)

Prognosis

केचित् क्लैब्ये त्वसाध्ये द्वे ध्वजभङ्गक्षयोद्भवे।
वदन्ति शेफसश्छेदाद्वृषणोत्पाटनेन च॥
मातापित्रोर्बीजदोषादशुभैश्चाकृतात्मनः।
गर्भस्थस्य यदा दोषाः प्राप्य रेतोवहाः सिराः॥
शोषयन्त्याशु तन्नाशाद्रेतश्चाप्युपहन्यते।
तत्र संपूर्णसर्वाङ्गः स भवत्यपुमान् पुमान्॥
एते त्वसाध्या व्याख्याताः सन्निपातसमुच्छ्रयात्।
चिकित्सितमतस्तूर्ध्वं समासव्यासतः शृणु॥

C.S.Ci 30/188-191

According to some physicians, impotency caused by *Dhvajabhaṅga* (morbidity of phallus) and *Kṣaya* (diminution of semen) are incurable. In this context, *Dhvajabhaṅga*, caused by amputation of the phallus and testicles is to be considered as incurable. The term *Kṣaya* in this context of incurability refers to the condition when because of the morbidities of the sperm of the father and ovum of the mother and because of the sinful actions of the past life, during the embryonic stage, the aggravated *Doṣās* afflict the channel carrying sperm, and make it atrophied. Because of this, (in the later part of life) the process of semen formation in the offspring is inhibited. Thus, the man though having full physical development becomes emaciated.

Different types of impotency described before which are caused by the simultaneous vitiation of all the three *Doṣās (Sannipāta)* are also incurable.

उपशय/पथ्य (Upaśaya/Pathya)

In cases of *Dhvajabhaṅga*, the following are considered to be beneficial: rice (*Śāli* and *Ṣaṣṭhi* types), wheat, *Masūra*, grams, butter, milk, *Surā*, *Sīdhu*, meats of duck, sparrow, hen, partridge, deer, rabbit and goat and fruit like grapes, date fruits, mango, *Jambū* and pomegranate. (B.R. 92/66-68)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

All the etiological factors.

वंध्यत्व (Vandhyatva)

वन्ध्यां नष्टार्तवम् विध्यात्। *Su. Utt. 38/10*

A women who has lost her *ārthava* is called as *Vandhya.*

यदा ह्यस्या शोणिताख्ये बीजे गर्भाशयस्ये वितर्कं बीजं प्रदुष्यति।
तदा वंध्यां जनयति॥ *A.Sm.Sha 2/46-47*

The congenital under development or deformity of female genital tract is responsible for *vandyatva.*

निदान (Nīdāna)

यदा स्त्रिया दोषप्रकोपणोक्तान्याः सेवमानया दोषाः.........। तदा स्त्र्याकृतिभूयिष्ठामास्त्रियं वार्ता नाम जयन्ति तां स्त्रीव्यापदमाचक्षते॥ *Ch. Sid. 12/30*

A. Vatakara food

अहारज (Āhāraja)

1. *Vātakara āhara* (intake of *vāta* aggravating factors)
2. *Taila, māmsa rahita āhāra* (consumption of food devoid of oil and meat) etc.

B. Vātakara activities

विहारज (Vihāraja)

Vātakara vīhāra like *rātrijāgaraṇa* (night vigil), *ati vyāyāma* (excessive exercise), *vegadhāraṇa* (suppression of natural urges) etc

Samprāpti (pathogenesis)

Due to consumption of etiological factors exacerbated *vāta* in a woman reaches the ovum and uterus but does not affect them entirely. She conceives and the foetus gets damaged in maternally derived organs. The child will be predominantly female shaped but she will be unable to conceive.

वन्द्ययस्यात् षद्प्रकारेण बाल्येनापि अथवा पुनः गर्भकोष्टस्य भंगाद्वा तथा धातुक्षयादपि जायते न च गर्भस्य संभूतिश्च कथाचन॥

Harita Samhita

- * *Balya* indicates the immaturity of female genital organs or delayed puberty.
- * *Garbha koṣa bhaṅga* : any pathology of uterus.

* *Dhātu kṣaya* : Due to *vyādhi* or *āhāra janya*

According to Caraka

सप्रजा अपि इति अवन्ध्या अपि सती कथं चिरेण गर्भ विन्दति ॥

Vandya - It means the absolute inability to become pregnant.

Aprajā - Infertility in which woman conceives after treatment.

Saprajā - It is a condition in which woman in her active reproductive age does not conceive after giving birth to one or more children.

Classification according to Caraka

Aprajā - Primary infertility- where no previous pregnancies reported.

Saprajā - Secondary infertility- Where there has been a previous documented pregnancy. It may be a live birth or failed pregnancy.

Classification according to Hārita

1. *Kākavandya* - Inability to conceive after one child.
2. *Anāpatya* - Primary sterility where the lady never conceives.
3. *Garbhasrāvi* - Infertility characterized by unsuccessful pregnancies due to repeated abortions.
4. *Mṛthavatsa* - Infertility characterized by unsuccessful pregnancies due to intrauterine death, stillbirths etc.
5. *Balakṣaya* - Infertility due to decrease in *dhātu* due to *dhātu kṣaya*, loss of strength. It indicates immaturity of female genital organs.
6. *Garbhakoṣa bhaṅga* - Infertility due to prolapse or injury to the uterus and loss of *dhātus*.

Classification According to Madhukoṣa

वन्ध्यानां नवधा प्रभेदा..........।

1. *Ādivandhyā pāpakarma nimittaja*
2. *Ratkaja*
3. *Vātaja*
4. *Pittaja*
5. *Kaphaja*
6. *Tridoṣaja*
7. *Bhūtaja*
8. *Daivaja*
9. *Abhicāra*

साध्यासाध्यता *(Sādhyāsādhyatā)*

1. *Vandhyā* in which the woman has congenital absence of uterus and *ārthava* is incurable.
2. All other types of *vandhyā* explained by *Hārita* are curable.
3. Infertility of women who had coitus before her menarche becomes pregnant with difficulty.

MCQs

1. Types of *Klaibya* as per *Charaka*

a. 6 b. 8

c. 4 d. 7

2. Types of *Vandyatva* as per *Harita*

a. 6 b. 8

c. 4 d. 7

3. Inability to conceive second child

a. *Anapatya*

b. *Kakavandya*

c. *Garbhakosabhanga*

d. *Apraja*

Answer

1.	c	2.	a	3.	b

CHAPTER

49

Sexual Dysfunction and Infertility

Male and Female Infertility

Infertility affects around 1 in 7 couples of reproductive age, often causing substantial psychological distress.

Causes of infertility in Women - Female factor-(35-40%)

A. May result from anovulation or from abnormalities of the reproductive tract that prevent fertilisation or embryonic implantation, most commonly damaged fallopian tubes from previous infection.

B. Ovulatory dysfunction
   - Polycystic ovarian syndrome
   - Hypergonadotrophic hypogonadism
   - Hypogonadotrophic hypogonadism

C. Tubular dysfunction
   - Pelvic inflammatory disease (chlamydia, gonorrhoea)
   - Previous sterilisation
   - Previous pelvic or abdominal surgery
   - Endometriosis

D. Cervical and/or uterine dysfunction
   - Congenital abnormalities
   - Treatment for cervical
   - Fibroids carcinoma
   - Asherman's syndrome

E. Unexplained or mixed factor (20-35%)

Causes of infertility in Men - Male factor (35-40%)

1. It may result from impaired sperm quality (e.g. reduced motility) or number.
2. Azoospermia or oligospermia is usually idiopathic, but may be a consequence of hypogonadism
3. Microdeletions of the Y chromosome are increasingly recognised as a cause of severely abnormal spermatogenesis.
4. In many couples, more than one factor causing subfertility is present, and in a substantial proportion no cause can be identified.
5. Reduced sperm quality or production
   - Y chromosome microdeletions
   - Hypergonadotrophic hypogonadism
   - Varicocele
   - Hypogonadotrophic hypogonadism
6. Tubular dysfunction
   - Varicocoele
   - Previous sexually transmitted infection (chlamydia, gonorrhoea)
   - Congenital abnormality of vas deferens/ epididymis
   - Previous vasectomy
7. Unexplained or mixed factor (20-35%)

Clinical assessment

1. A history of previous pregnancies, relevant infections and surgery is important in both men and women.
2. A sexual history must be explored sensitively as some couples have intercourse infrequently or only when they consider the woman to be ovulating, and psychosexual difficulties are common.
3. Irregular and/or infrequent menstrual periods are an indicator of anovulatory cycles in the woman, in which case causes such as PCOS should be considered.
4. In men, the testes should be examined to confirm that both are in the scrotum and to identify any structural abnormality, such as small size, absent vas deferens or the presence of a varicocoele.

Investigations

1. Investigations are generally performed after a couple has failed to conceive despite

unprotected intercourse for 12 months, unless there is an obvious abnormality (e.g. amenorrhoea).

2. The male partner should provide at least two fresh semen samples, over an interval of several weeks, for analysis of sperm count and quality.
3. In women with regular periods, ovulation can be confirmed by an elevated serum progesterone concentration on day 21 of the menstrual cycle.
4. In women with irregular periods, such as in PCOS, weekly urine samples can be collected over several months to detect any peaks in progesterone metabolites which indicate ovulation.
5. Transvaginal ultrasound can be used to assess uterine and ovarian anatomy.
6. Tubal patency may be examined at laparoscopy or by hysterosalpingography (HSG; a radio-opaque medium is injected into the uterus and should normally outline the fallopian tubes).
7. In vitro assessments of sperm survival in cervical mucus may be ascertained in cases of unexplained infertility but are rarely helpful.

Male hypogonadism

1. The clinical features of both hypo- and hypergonadotrophic hypogonadism include loss of libido, lethargy with muscle weakness, and decreased frequency of shaving.
2. Patients may also present with gynaecomastia, infertility, delayed puberty and/or anaemia of chronic disease.

Investigations

1. Male hypogonadism is confirmed by demonstrating a low serum testosterone level.
2. The distinction between hypo and hypergonadotrophic hypogonadism is by measurement of random LH and FSH.

Gynaecomastia

1. Gynaecomastia is the presence of glandular breast tissue in males.
2. Gynaecomastia results from an imbalance between androgen and oestrogen activity, which may reflect androgen deficiency or oestrogen excess.
3. The most common are physiological: for example, in the newborn baby (due to maternal and placental oestrogens), in pubertal boys (in whom oestradiol concentrations reach adult levels before testosterone) and in elderly men (due to decreasing testosterone concentrations).
4. Prolactin excess alone does not cause gynaecomastia
5. Hypogonadism, liver failure, oestrogen secreting tumour, Drugs (digoxin, cimetidine etc) etc may cause gynaecomastia.

Hirsutism

1. Hirsutism refers to the excessive growth of thick terminal hair in an androgen-dependent distribution in women (upper lip, chin, chest, back, lower abdomen, thigh, forearm) and is one of the most common presentations of endocrine disease.
2. It should be distinguished from hypertrichosis, which is generalised excessive growth of vellus hair..

Semen analysis, Semen Examination and Semen Counting Procedure

Sample

- This is preferred if the patient in the lab for the collection of the sample.
- Masturbation is preferred, and the entire collected semen should be submitted.
- The accepted volume is 2 to 5 mL.
- Collect the sample when the doctor or the technician should be available to evaluate the motility immediately.
- 2 to 3 days of sexual abstinence is preferred.
- Prolonged abstinence should be discouraged because of the sperm cells' quality, and especially their motility will decrease.
- The proper container is also important.
- To evaluate the vasectomy, the patient needs to ejaculate once or twice before the semen specimen collection day. This process will

clear the distal portion of the vas deference.

- Samples are unsatisfactory if the sample is obtained at the house by coitus interrupts or masturbation.
- In the house sample, instruct the patient to deliver it within one hour of collection.
- Instruct the patient to avoid cold and heat during transportation. The best is to keep the sample in the closed hands (in the fist).

Precautions

- Don't use condoms, particularly with spermicide.
- The specimen should be maintained at 37 °C during transport if brought from home and should be examined within 3 hours of collection.
- A sterile container is needed, and the sample should be collected at room temperature of 37 °C.
- Plastic containers are not recommended.
- Avoid extreme temperatures.
- The analysis should be done immediately when the semen is liquefied.
- Should be examined within 4 hours,
- The sample should be kept at 37 °C.
- Wait till liquefaction is complete for the examination.

Drugs that may decrease the count are

- Antineoplastic agents (nitrogen mustard, procarbazine, vincristine, and methotrexate).
- Direct the patient to avoid alcohol intake for several days before the semen collection.
- Instruct the patient to avoid sexual activity for 2 to 3 days before collecting the sample.
- Avoid prolonged abstinence because the quality of the sperms and their motility may be decreased.

Purpose of the Test (Indications)

- This is the workup of the infertile couple.
- This is done to evaluate the quality of sperms.
- To confirm the efficacy of vasectomy (postvasectomy).
- Done for the forensic studies.
- To get semen for artificial insemination

Pathophysiology

Definition of semen

- Seminal fluid is the secretion of prostate glands, two seminal vesicles, two Cowper glands, and testes serve as the vehicle for sperms transport and create the alkaline medium. It also provides nutrition. This serves as the medium which promotes the sperm's motility and survival.
- Semen (seminal fluid) consists of a combination of products of various male reproductive organs like testes, epididymis, seminal vesicle, prostate, bulbourethral, and urethral glands.
- The ejaculated semen is a viscous, yellow-grey fluid, which forms a firm gel-like clot immediately after the ejaculation.
- This clotted semen liquefies at room temperature (37 °C) within 5 to 60 minutes. If it requires more than one hour, that semen is considered abnormal.

Semen analysis includes

- Volume.
- Sperm count.
- Evaluation of the normal sperm.
- Evaluation of sperm motility.
- The pH of the semen.

Spermatogenesis occurs in the seminiferous tubules of the testes. These are matured in the epididymis

- Semen production is dependent upon the function of testes.
- These are stimulated by testosterone.
- The concentration of testosterone in the testes is 100 times more than the peripheral blood.
- This high concentration of testosterone is needed for spermatogenesis.
- LH stimulates Leydig's cells to produce testosterone.
- These are lined with 46 chromosome germ cells called spermatogonia.

- These cells undergo mitotic division and ultimately give rise to primary spermatocytes.
- Secondary spermatocytes give rise to spermatids, and these will differentiate into sperm or spermatozoa containing 23 chromosomes
- Semen production depends upon the function of the testis.
- Hypothalamus producing a gonadotropin-releasing hormone (GRH) in response to low testosterone levels.
- GRH stimulates the pituitary gland to produce the follicular stimulating hormone.
- Now the FSH stimulates the Sertoli cell, which is the location of sperm production.
- FSH stimulates the Sertoli cells to secrete activin, which stimulates sperma-togenesis.
- Besides, the Sertoli cells secrete inhibin, which inhibits FSH secretion from the pituitary gland.
- LH stimulates the Leydig cells, which produce testosterone. Testosterone then stimulates the seminiferous tubules to produce sperm.
- Spermatogenesis (in the seminiferous tubules) takes approximately 3 months.
- Maturation of the spermatogonia to mature spermatocytes takes approximately 75 days, through the epididymis.
- In the epididymis, the motility is acquired; it takes another 14 days.
- Spermatocytes develop from the Sertoli cells in the seminiferous tubules. They get matured in the epididymis and are ready for fertilization.
- Hemospermia when there are abundant RBCs in the ejaculate.

In primary gonadal failure

- FSH is raised.
- LH is raised.

In secondary gonadal failure

- FSH is decreased.
- LH is decreased.

Classification of spermatocytes dependent upon the sperm count

- Aspermia (azoospermia) when there is no sperm.
- Oligospermia when the count is < 20 million/mL.
- Asthenospermia is low sperm motility.
- Necrospermia - Normal counts of the sperm but are non-motile.

Semen Analysis Includes

1. Volume. Check when the sample is fresh.
2. Sperm count. This can be done when the semen is completely liquified.
3. Motility. This should be checked when the sample is fresh and repeat when it is liquefied.
4. Morphology. This can be better appreciated if the smear is stained.

Normal semen

1. The volume is 2 to 6 ml.
2. Color is gray to white or opalescent.
3. Liquefaction time normally 10 to 30 minutes at 37 °C. Some other says 15-20 min.
4. The volume normally is 1.5 to 5 mL.
5. pH varies between 7.7 to 8. Below, 7.0 usually has prostatic secretion.
6. Sperm count is 20 to 250 million/mL.
   - Normally a total count of more than 80 million/ml. But now, it is considered as > 20 million /mL as fertile semen.
7. Live spermatozoa of more than 50%.
   - >65% of the sperm does not take stain, and these are alive.
8. Sperm motility >50% at one hour. This is moderate to the strong forward motion.
   - Place the semen drop on the prewarmed slide at 37 °C.
   - Examine under the 40x and find the motile sperm.
   - Evaluate the sperm actively motile and sluggish one.
   - Normally >70% of sperms are motile after one hour when the sperms are kept at 37 °C.

9. The progressive motility score is 3 to 4.
10. Sperm morphology should be >30%. Normally normal forms are >70%, and immature forms are <4%.
    - Can stain the slide with the Papanicolaou method.
    - Count at least 200 sperms under oil emersion.
    - Abnormal forms are <30%.
    - Note immature sperms as well.
    - Also, note RBCs and polys.
11. Microscopy:
    - WBC is 4 to 6/HPF.
    - Epithelial cells are few.
    - RBCs are not found.
    - Aggregation may be seen, but very few.
    - Crystals are amorphous phosphate.
12. Stain the slide with eosine, which will be taken up by the dead sperms only.
13. Clumping or agglutination of the sperms should be reported, which indicates antibodies.
14. The fructose level is 150 to 600 mg/dL.
    - Low or nil fructose indicates an obstruction in the ejaculatory duct.
    - The spermatozoa utilize fructose.
15. Acid phosphatase = 100 to 300 mg/100 ml.
16. Citric acid = >3 mg/ml.
17. Zinc = >75 μ/ml.
18. Magnesium = >70 μ/ml.
19. Inositol = >1 mg/ml.
20. Glucosidase = >20 mU/ ejaculate.
21. Carnitine = >250 μ/ml.

Source 2 Normal semen

1. Volume = 2 to 5 mL
2. Liquefaction time = 20 to 30 minutes afte the collection.
3. pH = 7.12 to 8.0
4. Sperm count = 50 to 200 million/mL
5. Sperm motility = 60 to 80% actively motile.
6. Sperm morphology = 70 to 90% normal shaped

Normal semen (another source)

Characteristic features	Values per ejaculate
The volume of the semen per ejaculate	>2 mL
Sperm density	>20 million/mL
Sperm count	>40 million/mL
Motility	1. >50% forward progression 2. >25% rapid progression within 60 minutes of ejaculation
Morphology	>30% normal morphology
pH	7.2 to 8.0
Color (appearance)	Gray-white to yellow
Time of liquefaction	Within 30 minutes
Fructose level	>1200 μ/mL
Acid phosphatase	100 to 300 mg/mL
Citric acid	>3 mg/mL
Zinc	>75 μ/mL
Magnesium	>70 μ/mL
Inositol	>1 mg/mL
Carnitine	>250 μ/mL
Prostaglandins (PGE1 and PGE2)	30 to 200 μ/mL
Glucosidase	>20 mU per ejaculate
Glycerylphosphory-lcholine	>650 μ/mL

Pathological Semen

1. The color is brown to red. or may be yellow and turbid.
2. Liquefaction time is >60 minutes.
3. Suppose the volume is <1.5 ml. This maybe <2 ml or >5 ml.
4. pH is <7.2.
5. Total count <20 million /mL.
6. Live sperm <35 %.
7. Motility <50 % after 2 hours.

8. The progressive motility score is 0 to 1.
9. Morphology >30 % abnormal forms.
10. Fructose is decreased.

Difference between the normal and abnormal semen:

Parameter	Normal semen	Pathological semen
Coagulation	Coagulates	Delayed
Liquefaction	Complete in 10 to 30 minutes	Delayed
pH	7.2 to 7.8	
Volume	2 to 6 mL	<1.5 mL
Sperm density x 106/mL	>20	<10
Total sperm count x106/mL/ per ejaculate	>80	<20
Progressive motility score (after 2 to 4 hours of ejaculate)	3 to 4	0 to 1
Live spermatozoa	≥ 50%	<35%
One hour after ejaculate	≥ 70%	
After 3 hours of ejaculate	≥ 60%	
After 4 hours of ejaculate	>50%	<35%
Normal spermatozoa	≥ 60%	<35%
Defective heads	<35%	>60%
Defective mid-piece	≥ 20%	>25%
Defective tails	≥ 20%	>25%
Immature forms	<4%	

The first method to count Sperms

1. **Sperm count solution:**
   - Formalin = 1 mL
   - Sodium bicarbonate = 5 grams
   - Distilled water = 100 mL
   - Mix well the above ingredients.
   - Add all the above ingredients into 100 mL distilled water.
2. **Procedure for counting the sperms:**
   - Take 0.02 ml semen
   - Take 0.38 mL above solution (diluting fluid).
   - Mix well above semen and diluting solution.
   - Now load the Neubauer chamber.
   - Count the large two squares.
3. **Important** that moves the microscope up and down to count the sperm at a different level.
4. **Calculation:** Count in the large two square x100,000 = count in million /mL

Formula to calculate the sperm count

Sperm count = Number of sperms in 2 large square (2 sq. mm) x 100,000=Count in million/mL

Counting procedure

- Improved Neubauer chamber (Haemocytometer) is used for counting
- The diluted semen is carefully mixed and the chamber is charged using a Pasteur pipette.
- The chamber is then examined by using x 10 objective of microscope.
- Sperms are counted in the four large corners and one large central square.

The second method to count sperms in the semen:

1. Dilute the semen with water or above sodium bicarbonate solution.
2. Take 1:20 dilution of the semen.
3. Count the five RBC counting squares (4 at the corner and one at the center).
4. **Calculations:** suppose the sperms are 50 in all these 5 RBC square.

   1.50 x 1,000,000 = 50,000,000/mL (million/mL)

Semen : Microscopic Analysis

- Sperm count
- NV = 20-160 million / mL
- Make 1 to 20 dilution with sodium

bicarbonate and fromalin, count 5 small squares (within the center large square) of the Neubauer hemacytometer.

To calculate % motility

1. Count at least 200 motile or non-motile sperms in 5 different microscopic fields. % of the nonmotile sperm are calculated as follows:

Formula to calculate the sperm motility

$$\text{Motility} = \frac{\text{Total sperms - Nonmotile sperms \%}}{\text{Total sperms}} \times 100$$

Normally, 50% or more sperms are motile

Sperm viability

- The ejaculated sperm in the semen remain viable for several days in the female genital tract. Fertilization can occur as long as the sperms are alive, and these can survive for 5 days.
- The sperms can be preserved for decades when the semen is freezed at -20 °C.
- Sperm viability is needed when the progressively motile sperms are less in number.

Method to assess the viability

1. Take one drop of the semen.
2. Add an equal volume of vital stain (trypan blue).
3. Cover with the cover glass.
4. Keep for a few minutes, but not more than 5 minutes.
5. Count 100 cells, including viable sperm (not stained) and dead sperm (take stain).
6. This will give you the count of viable sperms.

Forensic Value of Semen Analysis

1. **Acid phosphatase** is in high concentration in the semen.
2. It is suggested that a swab from the suspected case of rape is taken, preserve in 2.5 mL of a protective broth, and store at 4 °C or room temperature.
   - A sample stored in this way will keep acid phosphatase activity for one month.
3. In the sample with thymolphthalein as the substrate, it was found that vaginal acid phosphatase in noncoital women is <10 U/L.
   - There is a noncoital level if the vaginal sample is taken after 4 days of intercourse.
4. The sampling swab can be kept in 1 mL of normal saline, but these samples are freezed and thaw at 2 to 4 °C for 24 hours before the assay.
5. **Creatinine phosphokinase** (CPK) is a high level in the semen. This can also be used for medicolegal purposes.

Importance of the Semen Analysis

1. That one sample cannot confirm the infertile couple because of variation in the count of semen.
2. To repeat one more sample. If possible, take the third sample at an interval of 3 weeks apart.
3. In case of vasectomy, no sperms should be seen in the ejaculate.

Work up for the infertile male

1. Get the report of the previous semen analysis.
2. Advise antispermatozoal antibody.
3. Advise luteinizing hormone (LH) and Follicle-stimulating hormone (FSH). These hormones will give the effect of the pituitary gland on gonadal function and spermatogenesis.
4. Sims-Huhner test. In this test, a postcoital cervical mucus smear is taken. To see the sperm mobility penetration in the mucus and maintenance of the mobility.

Drugs that may decrease the Count are

1. Cancer chemotherapy (Vincristine, methotrexate, procarbazine, and nitrogen mustard).
2. Estrogen therapy.
3. Cimetidine.
4. Methyltestosterone.

Important Note

- Single semen analysis is not conclusive because the sperm count varies from day today.
- A semen analysis should be done twice or thrice for the best result

Sperm Antibody, Anti Sperm-antibody, Anti Spermatozoal antibody

Sample

1. This is done on the fresh semen of the patient and sample preferred by masturbation.
2. First, urinate and then clean and wash the penis before collecting the sample.
3. Keep the sample at body temperature if collected at home.
4. The sample cannot be collected by sexual intercourse because there will be contamination by vaginal secretion.
5. Take the blood of the male and female for serum preparation.
6. Collect cervical mucus from the female.

Purpose of the Test (Indications)

1. This is a screening test for an infertile couple.
2. This detects antibodies against the spermatozoa.

Pathophysiology

1. Sperm antibodies are directed against the sperm antigen.
2. Antisperm antibodies are found in:
   - Men with the blocked efferent duct.
   - Men with a vasectomy show a 30 to 70% antibody.
3. Mechanism of antibody formation.
   - Sperm from the blocked ducts gives rise to autoantibody formation as the immune system recognizes these as forgein.These antibodies are agglutinin or cytotoxic.
4. The antisperm antibody of IgA type is against the tail of sperm, and they inhibit the motility and decrease the penetration of cervical mucus.
5. Sperm antibody of IgG type blocks the sperm-ovum fusion.
6. A high titer of sperm antibodies is strong evidence of infertility.
7. Sperm antibodies are found in 20% of the female. Their presence in the female significance is not clear because some of these ladies may become pregnant.
8. In some studies :
   1.Women with primary infertility are 75% positive for sperm agglutinin antibodies.
   2.11 to 15% of pregnant ladies show the same level of sperm antibodies.

Normal

- Negative.

Anti-Sperm Antibodies are seen in:

1. Blockage of testicular efferent ducts.
2. After the vasectomy, almost every male has an antisperm antibody. After the reversal of the vasectomy antisperm, the antibody disappears.
3. Infertility. This may be seen in both males and females.

Treatment

For to treat antisperm antibodies, the possibilities are:

1. Use of the condom.
2. Sperms are processed to remove the antibodies.
3. Immunosuppressive therapy can be given both in males and females.
4. Invitro fertilization (IVF) can be tried.

Differential diagnosis of infertility and sexual dysfunction

S.No.	Infertility	Sexual dysfunction
1	Infertility is the failure to conceive (regardless of cause) after 1 year of unprotected intercourse.	Disturbed sexual intercourse & pleasure
2	This condition affects approximately 10-15% of reproductive-aged couples.	Sexual dysfunction can be any problems that prevent a person or couple from experiencing satisfaction from sexual activity. Some 43% of women and 31% of men report some degree of sexual dysfunction.
3	**Female factors** that affect fertility include the following categories: **Cervical:** Stenosis or abnormalities of the mucus-sperm interaction **Uterine:** Congenital or acquired defects; may affect endometrium or myometrium; may be associated with primary infertility or with pregnancy wastage and premature delivery **Ovarian:** Alteration in the frequency and duration of the menstrual cycle-Failure to ovulate is the most common infertility problem **Tubal:** Abnormalities or damage to the fallopian tube; may be congenital or acquired **Peritoneal:** Anatomic defects or physiologic dysfunctions (eg, infection, adhesions, adnexal masses)	**Female factors** **Desire disorders:** lack of sexual desire or interest in sex. **Arousal disorders:** inability to become physically aroused or excited during sexual activity. **Orgasm disorders:** delay or absence of orgasm (climax). **Pain disorders:** pain during intercourse. **Lack of vaginal lubrication:** Inadequate vaginal lubrication before and during intercourse. **Vaginal muscles:** Inability to relax the vaginal muscles enough to allow intercourse.
4	**Male factors:** **Pre-testicular:** Congenital or acquired diseases of the hypothalamus, pituitary, or peripheral organs that alter the hypothalamic-pituitary axis **Testicular:** Genetic or nongenetic **Post-testicular:** Congenital or acquired factors that disrupt normal transport of sperm through the ductal system	**Male factors:** • Inability to achieve or maintain an erection (hard penis) suitable for intercourse (erectile dysfunction). • Absent or delayed ejaculation despite enough sexual stimulation (retarded ejaculation). • Inability to control the timing of ejaculation (early, or premature ejaculation).

MCQs

1. Infertility is when a couple fails to conceive after
   a. 4 years of unprotected sex
   b. 3 years of unprotected sex
   c. 2 years of unprotected sex
   d. 1 year of unprotected sex
2. In women, this sexually transmitted disease can result in infertility
   a. Human papillomavirus (HPV)
   b. Pelvic inflammatory disease
   c. Genital herpes
   d. All of the above

Answer

1.	d	2.	b

CHAPTER

50

Unmada and Apasmara

उन्माद निदान (Unmāda Nidāna)

Introduction

Unmāda or insanity is of six varieities, as follows. Caused by excessive aggravation of either *vāyu* or *pitta* or *kapha*; Caused by the excessive aggravation of all the three *doṣas* simultaneously; Caused by mental grief; and Caused by poisons. *Vātika unmāda*, *Paittika unmāda*, *Kapahaja unmāda*, *Sannipātika unmāda*, *Viṣajanya unmāda*, *Bhūtottha unmāda*, *Devonmāda*, *Rākṣasonmāda*, *Gandhar-vonmāda*, *Yakṣonmāda*, *Pretonmāda*, *Bhujangonmāda*, *Brahmarā-kṣasonmāda* and *Piśāconmāda*. In the first stage of the disease, when the condition is little moderate in nature, it is called *mada*. Various types of *unmāda* (insanity) are caused by the following factors: Intake of mutually contradictory food; Intake of polluted food; Intake of unclean food; Showing disrespect to the gods, preceptors and *dvijas* (people of upper three castes among Hindus); Affliction of the mind by excessive fear of exhilaration; and Improper postures. By the above-mentioned factors, *doṣa* get aggravated in a person with a weak mind, and they afflict the heart, which is the location of intellect. As a consequence of this, *doṣa* pervade through *manovaha srotas* (channels of the mind) and thereafter, they cause *pramoha* (impairment of functioning) of the *cetas* (mind, including intellect). The signs and symptoms of *unmāda* (insanity) in general, are as follows: impairment of intellect; instability of mind; anxious look; impatience; incoherent speech; and feeling of emptiness in the heart.

उन्माद निरुक्ति (Unmāda Nirukti)

उन्मादं पुनर्मनोबुद्धिसंज्ञाज्ञानस्मृतिभक्तिशीलचेष्टाचारविभ्रमं विद्यात्॥ *C.S.Ni. 7/5*

Characteristic features of *Unmāda* are perversion of mind, intellect, consciousness, knowledge, memory, desire, manners, behaviour & conduct.

उन्माद हेतु एवं संप्राप्ति (Unmāda Hetu evam Samprāpti)

तत्र दोषनिमित्ताश्चत्वारः पुरुषाणामेवंविधानां क्षिप्रमभिनिर्वर्तन्ते; तद्यथाभीरूणामुपक्लिष्टसत्वानामुत्सन्नदोषाणां समलविकृतोपहितान्यनुचितान्याहारजातानि वैषम्ययुक्तेनोपयोगविधिनोपयुञ्जानानां तन्त्रप्रयोगमपि विषममाचरतामन्याश्च शरीरचेष्टा विषमाः समाचरतामत्युपक्षीणदेहानां व्याधिवेगसमुद्भमितानामुपहतमनसां वा कामक्रोधलोभहर्षभयामोहायासशोकचिन्तोद्वेगादिभिर्भूयोऽभिघाताभ्याहतानां वा मनस्युपहते बुद्धौ च प्रचलितायामभ्युदीर्णा दोषाः प्रकुपित्ता हृदयमुपसृत्य मनोवहानि स्त्रोतांस्यावृत्य जनयन्त्युन्मादम्॥ *C.S.Ni. 7/4*

विरुद्धदुष्टाशुचिभोजनानि प्रधर्षणं देवगुरुद्विजानाम् ।
उन्मादहेतुर्भयहर्षपूर्वो मनोऽभिघातो विषमाश्च चेष्टाः॥
C.S.Ci. 9/4

तैरल्पसत्वस्य मलाः प्रदुष्टा बुद्धेर्निवासं हृदयं प्रदूस्य ।
स्त्रोतांस्यधिष्ठाय मनोवहानि प्रमोहयन्त्याशु नरस्य चेतः ॥
C.S.Ci. 9/5

Four varieties of *Unmāda* caused by the vitiation of *Doṣās* manifest themselves instantantenously in the following persons : One who is timid; dominancy of *Rajas* & *Tamo Doṣa*; aggravation and vitiation of *Doṣās;* consumption of unclean, unwholesome & damaged food substances; one who does not observe rules & regulations of dietetics; improper application of tantric practices; abnormal postures; extreme emaciation; who is suffering from chronic diseases; whose mind is inflicted by passion, anger, greed, exhilaration, fear, attachment, exertion, grief, anxiety, excitement etc; one who is frequently subjected to physical assault & insulting gods, preceptors & *Dvijās*.

उन्माद संप्राप्ति (Unmāda Samprāpti)

Due to above etiological factors mind gets damaged & intellect becomes unstable, in this state the aggravated *Doṣās* spreading to the heart & obstruct the *Manovaha Srotas* as a result *Unmāda* manifests.

मदयन्त्युद्धता दोषा यस्मादुन्मार्गमाश्रिताः ।
मानसोऽयमतो व्याधिरुन्माद इति कीर्त्तितः ॥

S.S.Utt. 62/3

Excessively aggravated *Doṣās* getting localised in the upper paths cause abnormality in the mind, resulting in the manifestion of *Unmāda*, which is a disease of the *Manas*.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Doṣās* both *Śārīrika* and *Mānasika (Rajas & Tamas,)*
↓
Doṣās spreading to the heart and *Manovaha Srotas* via *Dhamani*
↓
localised in the upper paths cause abnormality in the mind
↓
Obstruct the *Manovaha Srotas*
↓
Favours the localisation of *doṣās & dūṣyās* in the *Manovaha Srotas*
↓
Unmāda

उन्माद संप्राप्ति घटक (Samprāpti Ghaṭaka of Unmāda)

Doṣa	: *Śārīrika* *Doṣa* → *Tridoṣaja* *Pitta* → *Sādhaka* *Kapha* → *Tarpaka* *Vāta* → *Udāna* *Mānasika Doṣa* → *Rajas & Tamas*
Dūṣya	: *Dhātu* → *Rasa* *Upadhātu* → *Śārīrika Mala*→ *Dhātumala*→
Agni	: *Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi	: *Mandatā*
Āma	: *Āgnijanya*
Srotas	: *Manovaha*
Srotoduṣṭi	: *Saṅga, Atipravṛtti, Vimārgagamana*
Udbhavasthāna	: *Hṛdaya*
Vyaktasthāna	: *Sarva Śarīra*
Sancārasthāna	: *Manovaha Sroto Avayava*
Adhiṣṭhāna	: *Manas, Buddhi*
Svabhāva	: *Cirakāri & Āśukāri*
Prabhāva	: *Kṛcchrasādhya*
Rogamārga	: *Madhyama*
Roga bheda	: 5 (*Vātaja, Pittaja, Kaphaja, Sannipātaja & Āgantuja) (Caraka)* 6 (*Vātaja, Pittaja, Kaphaja, Sannipātaja, Mānasaja & Madaja) (Suśruta)*

उन्माद पूर्वरूप (Unmāda Pūrvarupa)

तस्येमानि पूर्वरूपाणि: तद्यथा-शिरसः शून्यता, चक्षुषोराकुलता, स्वनः कर्णयोः उच्छ्वऽसस्याधिक्यम्, आस्यसंस्त्रवणम्, अनन्नाभिलाषारोचकाविपाकाः, हृद्ग्रहः, ध्यानायाससंमोहोद्वेगाश्चास्थाने, सततं लोमहर्षः, ज्वरश्चाभीक्ष्णम्, उन्मत्तचित्तत्वम्, उदर्दित्वम्, उर्दिताकृतिकरणं च व्याधेः, स्वप्ने चाभीक्ष्णं दर्शनं भ्रान्तचलितानवस्थितानां च तिलपीडकचक्राधिरोहणं वातुकुण्डलिकाभिश्चोन्मथनं निमज्जनं च कलुषाणामम्भसामावर्ते चुक्षषोश्चापसंर्पणमिति (दोषनिमित्तानामुन्मादानां पूर्वरूपाणि भवन्ति)॥

C.S.Ni. 7/6

मोहोद्वेगौ स्वनः श्रोत्रे गात्राणामपकर्षणम् ।
अत्युत्साहोऽरुचिश्चान्ने स्वप्ने कलुषभोजनम् ॥
वायुनोन्मथनञ्चापि भ्रमश्चक्रगतस्य वा ।
यस्य स्यादचिरेणैव उन्मादं सोऽधिगच्छति ॥

S.S.Utt. 62/6-7

Premonitory signs & symptoms of *Unmāda* are as follows : Feeling of emptiness in head, excitement in eyes, dizziness in ears, increased inspiration, excessive salivation, aversion to food, anorexia, indigestion, stiffness in cardiac region, meditation, exhaustion, bewilderment, excitement in improper situations, continuous horripilation, frequent fever, fickle mindedness, urticarial patches, facial expression as if affected by facial paralysis, frequent appearance of inauspicious objects that are wandering, moving & unstable, riding over the wheel of an oil press, being charted by whirl winds, sinking in a whirl of turbid waters, divergence of eyes; delusions; emotional bouts; debility of the body; great enthusiasm; dreams as though eating dirty foods; twisting pain in the body & giddiness as though turning on a wheel.

सामान्य रूप (Sāmānya Rupa)

धीविभ्रमः सत्वपरिप्लवश्च पर्याकुला दृष्टिरधीरता च ।
अबद्धवाक्त्वं हृदयं च शून्यं सामान्यमुन्मादगदस्य लिङ्गम्॥
C.S.Ci. 9/6

General Signs & symptoms of *Unmāda* are as follows:– Perverted intellect, fickleness of mind, unsteadiness of the vision, impatience, incoherent speech and feeling of emptiness in heart.

स मूढचेता च सुखं न दुःखं नाचारधर्मौ कुत एव शान्तिम् ।
विन्दत्यपास्तस्मृतिबुद्धिसंज्ञो भ्रमत्ययं चेत इतस्ततश्च ॥
C.S.Ci. 9/7

समुद्भ्रमं बुद्धिमनःस्मृतीनामुन्मादमागन्तुनिजोत्थमाहुः । तस्योद्भवं
पञ्चविधं पृथक् तु वक्ष्यामि लिङ्गानि चिकित्सितं च ॥
C.S.Ci. 9/8

Due to derangement of mind *Unmāda* patients does not experience pleasure, sorrow, ethics & religion, that's why these patients loses peace of mind altogether & become devoid of memory, intellect & recognition and patient moves restlessly here & there.

Unmāda is the perversion of intellect, mind & memory. It is classified into *Āgantuja* & *Nija*.

रोग भेद (Roga Bheda)

इह खलु पञ्चोन्मादा भवन्ति; तद्यथा-वातपित्तकफसन्निपातागन्तुनिमित्ताः॥ *C.S.Ni. 7/3*

एकैकशः समस्तैश्च दोषैरत्यर्थमूर्च्छितैः ।
मानसेन च दुःखेन स पञ्चविध उच्यते ॥
विषाद्भवति षष्ठश्च यथास्वं तत्र भेषजम् ।
स चाप्रवृद्धस्तरुणो मदसंज्ञां बिभर्त्ति च ॥
S.S.Utt. 62/4-5

Caraka classified *Unmāda* into five types namely *Vātaja, Pittaja, Kaphaja, Sannipātaja* & *Āgantuja*. *Suśruta* Classified *Unmāda* into 6 varieties namely *Vātaja, Pittaja, Kaphaja, Sannipātaja. Mānasaja* and *Viṣaja*.

विशेष रूप (Viśeṣa Rupa)

वातज उन्माद (Vātaja Unmāda)

परिसरणमजस्त्रम्, अक्षिभ्रुवौष्ठांसहन्वग्रहस्तपादाङ्गविक्षेपणमकस्मात्, सततमनियतानां च गिरामुत्सर्ग, फेनागमनमास्यात्, अभीक्ष्णं स्मितहसितनृत्यगीतवादित्रसंप्रयोगाश्चास्थाने, वीणावंशशङ्खशम्यातालशब्दानुकरणमसाम्ना, यानमयानैः, अलङ्करणमनलङ्कारिकैर्द्रव्यैः, लोभश्चाभ्यवहार्येष्वलब्धेषु, लब्धेषु चावमानस्तीव्रमात्सर्यं च, कार्श्यं, पारुष्यम्, उत्पिण्डितारुणाक्षता, वातोशयविपर्यासादनुपशयता च; इति वातोन्मादलिङ्गानि भवन्ति।
C.S.Ni. 7/7(1)

रूक्षच्छविः परुषवाग् धमनीततो वा शीतातुरः कृशतनुः स्फुरिताङ्गसन्धिः । आस्फोटयत्यटति गायति नृत्यशीलो विक्रोशति भ्रमति चाप्यनिलप्रकोपात् ॥ *S.S.Utt. 62/8*

Characteristic features of *Vātaja Unmāda* are as follows : Constant wandering, sudden movement of eyes, eyebrows, lips, shoulder, jaws, fore-arm & legs; continous incoherent speech, froath comes out from mouth; patient always smiling, laughing, weeping, dancing, shivering, singing & playing with musical instrument in appropriate situations; imitating the sounds of lute, flute, conch, *Śamya* (symbol played by right hand) and *Tāla* (symbol played by left hand) loudly, riding over undesirable vehicles; decorating with non adorning materials, longing for nonavailable eatables and disliking for food substances & strong dislike for available food items; emaciation; roughness; projected & reddish eyes; *Vāta* aggravating factors enhance the disease condition; rough speech, prominent blood vessels; desirous of cold and throbbing in body parts.

पित्तज उन्माद (Pittaja Unmāda)

अमर्षः क्रोधः, संरम्भश्चास्थाने, अस्त्रलोष्ट्रकशाकाष्ठमुष्टिभिरभिहननं स्वेषां परेषां वा, अभिद्रवणं, प्रच्छायशीतोदकान्नाभिलाषः, संतापश्चातिवेलं, ताम्रहरितहारिद्रसंरब्धाक्षता, पित्तोपशयविपर्यासादनुपशयता च; इति पित्तोन्मादलिङ्गानि भवन्ति। *C.S.Ni. 7/7(2)*

तृट्स्वेददाहबहुलो बहुभुग्विनिद्रश्छायाहिमानिलजलान्तविहारसेवी। तीक्ष्णो हिमाम्बुनिचयेऽपि स वह्निशङ्की पित्ताद्दिवा नभसि पश्यति तारकाश्च ॥ *S.S.Utt. 62/6*

Characteristic features of *Pittaja Unmāda* are as follows : Irritation, anger, inopportunate excitement, inflicting injury to own people or others with weapons, brick bats, whips, wooden sticks, fests, fleeing, desire for shade, cold water & food, excessive anguish for long; coppery, greenish, yellowish & congested eyes; aggravation of the condition by *Pitta* aggravating factors; thirst, sweating, burning sensation, eats more, sleeplessness, vicinity of water reservoirs, violent, suscpects the presence of fire even in ice & water and sees stars in the sky even during day.

कफज उन्माद (Kaphaja Unmāda)

स्थानमेकदेशे, तूष्णींभावः, अल्पशश्चङ्क्रमणं, लालाशिङ्घाणकस्त्रवणम्, अनन्नाभिलाषः, रहस्कामता, बीभत्सत्वं, शौचद्वेषः, स्वप्ननित्यता, श्वयथुराननै, शुक्लस्तिमितमलोपदिग्धाक्षत्वं, श्लेष्मोपशयविपर्यासादनुपशयता च; इति श्लेष्मोन्मादलिङ्गानि भवन्ति। *C.S.Ni. 7/7(3)*

Characteristic features of *Kaphaja Unmāda* are as follows : Standing in one place, observing silence, little movements, discharge of saliva & nasal secretions, aversion towards food, love for solitude, loathsomeness, dislike for cleanliness, always sleeps, swelling on face, white & timid eyes with excreta adhered to them, aggravation of the condition by *Kapha* aggravating factors, vomiting, weakness of digestive fire, debility to the body, anorexia, cough, desire the company of women in solitude, poor intellect & talk, talk less, eats less, desires hot things; symptoms increase after food & at night.

सन्निपातज उन्माद (Sannipātaja Unmāda)

त्रिदोषलिङ्गसन्निपाते तु सान्निपातिकं विद्यात्; तमसाध्यमाचक्षते कुशलाः। *C.S.Ni. 7/7*

सर्वात्मके पवनपित्तकफा यथास्वं संहर्षिता इव च लिङ्गमुदीरयन्ति ॥ *S.S.Utt. 62/11*

Characteristic features of *Sannipātaja Unmāda* are as follows : Presence of all the signs & symptoms of all the three *Doṣa* are manifested in aggravated form and appear as though competing with one another. It is incurable.

आगन्तुज उन्माद (Āgantuja Unmāda)

यस्तु दोषनिमित्तेभ्य उन्मादेभ्यः समुत्थानपूर्वरूपलिङ्गवेदनोपशयविशेषसमन्वितो भवत्युन्मादस्तमागन्तुकमाचक्षते। केचित् पुनः पूर्वकृतं कर्माप्रशस्तमिच्छन्ति तस्य निमित्तम्। तस्य च हेतुः प्रज्ञापराध एवेति भगवान् पुनर्वसुरात्रेयः। प्रज्ञापराधाद्ध्ययं देवर्षिपितृगन्धर्वयक्षराक्षसपिशाचगुरुवृद्धसिद्धाचार्यपूज्यानवमत्याहितान्याचरति, अन्यद्वा किंचिदेवंविधं कर्माप्रशस्तमारभते; तमात्मना हतमुपघ्नन्तो देवादयः कुर्वन्त्युन्मत्तम्॥ *C.S.Ni. 7/10*

देवर्षिगन्धर्वपिशाचयक्षरक्षःपितृणामभिधर्षणानि ।
आगन्तुहेतुर्नियमव्रतादि मिथ्याकृतं कर्म च पूर्वदेहे ॥
C.S.Ci. 9/16

Āgantuja Unmāda is different from the *Doṣic* varieties of *Unmāda* in terms of etiology, prodromal signs & symptoms, signs & symptoms, pain and homologation. Some people say that it manifests due to sinful activities of past life. *Punarvasu Ātreya* considers that *Prajñāparādha* as the causative factors for this disease. Due to intellectual blasphemy the person disregards the gods, sages, ancestors, *Gandharvās, Yakṣās, Rākṣās, Piśācas,* preceptors, elders, accomplished persons, teachers & other respectable persons. Person behaves unwholesomely or indulges in inauspicious activities. Person suffers from this *Unmāda* due to infliction by his own inasuspicious activities associated with infliction by gods etc.

आगन्तुज उन्माद पूर्वरूप (Āgantuja Unmāda Pūrvarupa)

तत्र देवादिप्रकोपनिमित्तेनागन्तुकोन्मादेन पुरस्कृतस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा: देवगोब्राह्मणतपस्विनां हिंसारुचित्वं, कोपनत्वं, नृशंसाभिप्रायता, अरतिः, ओजोवर्णच्छायाबलवपुषामुपतप्तिः स्वप्ने च देवादिभिरभिभर्त्सनं प्रवर्तनं चेति; ततोऽनन्तरमुन्मादाभिनिर्वृत्तिः॥ *C.S.Ni. 7/11*

Premonitory signs & symptoms of *Āgantuja Unmāda* caused by the wrath of gods etc. are as follows : Desire for inflicting voilence on gods, cows, *Brāhmins* and ascetics; anger; indulging in cruel disposition; uneasiness; impairment of *Ojas;* complexion, lustre, strength; abuse and incitement by the gods etc. in dreams followed by development of *Āgantuja Unmāda.*

Triggering and risk factors for Āgantuja Unmāda

उन्मादयिष्यतामपि खलु देवर्षिपितृगन्धर्वयक्षराक्षसपिशाचानां गुरुवृद्धसिद्धानां एष्वन्तरेष्वभिगमनीयाः पुरुषा भवन्ति; तद्यथा- पापस्य कर्मणः समारम्भे, पूर्वकृतस्य वा कर्मणः परिणामकाले, एकस्य वा शून्यगृहवासे चतुष्पथाधिष्ठाने वा, सन्ध्यावेलायामप्रयतभावे वा पर्वसन्धिषु वा मिथुनीभावे, रजस्वलाभिगमने वा, विगुणे वाऽध्ययनबलिमङ्गलहोमप्रयोगे, नियमव्रतब्रह्मचर्यभङ्गे वा, महाहवेवा देशकुलपुरविनाशे वा, महाग्रहोपगमने वा, स्त्रिया वा प्रजननकाले, विविधभूताशुभाशुचिस्पर्शने वा, वमनविरेचनरुधिरस्रावे, अशुचेरप्रयतस्य वा चेत्यदेवायतनाभिगमने वा, मांसमधुतिलगुडमद्योच्छिष्टे वा, धर्माख्यानव्यतिक्रमे वा, अन्यस्य वा कर्मणोऽप्रशस्तस्यारम्भे, इत्यभिघातकाला व्याख्याता भवन्ति॥ *C.S.Ni. 7/14*

Persons fall victims to the attack of *Unmāda* caused by gods, sages, ancestors, *Gandharvās, Yakṣās, Rākṣasās, Piśācās,* preceptors, elders and accompanied persons in the following circumstances :

In the beginning of sinful acts; at the time of maturation of the past deed; residing in a deserted house or going to cross roads alone; sexual intercourse in the new moon & the full moon days, sexual intercourse with a women during menses; recitation of scriptures, religious offerings, auspicious rites and ablations in improper manner; dishonouring a vow, religious duty and celibacy; great wars, destruction of country, family & towns; onset of inauspicious planets in the sky at the time of delivery of women; inauspicious & unclean touch of various creatures; during emesis, purgation & blood letting; visiting a *Caitya* (sacred tree) or religious places while unclean & carelessly; resorting to the remnants of meat, honey, sesamum, jaggery and alcohol, in naked condition while naked; visiting cities, towns, cross roads, corporation, gardens, cremation grounds, and slaughter houses at night; insulting *Brāhmans,* preceptors, gods, ascetics and other respectable persons; misinterpretation & religious scriptures and performance of any other inauspicious acts.

आगन्तुज उन्माद लक्षण/भूतोन्माद लक्षण (Āgantuja Unmāda Lakṣaṇa/Bhūtonmāda Lakṣaṇa)

अमर्त्यवाग्विक्रमवीर्यचेष्टो ज्ञानादिविज्ञानबलादिभिर्यः ।
उन्मादकालोऽनियतश्च यस्य भूतोत्थमुन्मादमुदाहरेत्तम् ॥
C.S.Ci. 9/17

तस्येमानि रूपाणि भवन्ति; तद्यथा-अत्यात्मबलवीर्यपौरुषपराक्रमग्रहणधारणस्मरणज्ञानवचनविज्ञानानि, अनियतश्चोन्मादकालः॥ *C.S.Ni. 7/13*

General signs & symptoms of *Āgantuja* or *Bhūtonmāda* are as follows : Supernatural strength, energy, manliness, enthusiasm, power of understanding & retention, memory, spiritual as well as artistic knowledge & power of speech in the patient himself and activities manifested as a result of super natural knowledge & uncertain time of paroxysms.

आगन्तुज उन्माद संप्राप्ति (Āgantuja Unmāda Samprāpti)

The causative agents of the *Āgantuja Unmāda* initiate their actions such as gods produce insanity by their vision, preceptors, elders, accomplished persons & the great sages by cursing; ancestors by exposing themselves; *Gandharvās* by their touch, *Yakṣās* by entering *Rākṣasās* by making the patient to smell the odour of their body; *Piśācās* by riding & driving their victims.

आगन्तुज उन्माद विशिष्ठ लक्षण (Āgantuja Unmāda Viśiṣṭa Lakṣaṇa)

तद्यथा–सौम्यदृष्टिं गम्भीरमधृष्यमकोपनमस्वप्नभोजनाभिलाषिणमल्पस्वेदमूत्रपुरीषवातं शुभगन्धं फुल्लपद्मवदनमिति देवोन्मत्तं विद्यात्, गुरुवृद्धसिद्धर्षीणामभिशापाभिचाराभिध्यानानुरूप- चेष्टाहारव्याहारं तैरुन्मत्तं विद्यात्; अप्रसन्नदृष्टिमपश्यन्तं निद्रालुं प्रतिहतवाचमनन्नाभिलाषमरोचकाविपाकपरीतं च पितृभिरुन्मत्तं विद्यात्; (चण्डं साहसिकं तीक्ष्णं गम्भीरमधृष्यं) मुखवाद्यनृत्यगीतान्नपानस्नानमाल्यधूपगन्धरतिं रक्तवस्त्रबलिकर्महास्यकथानुयोगप्रियं शुभगन्धं च गन्धर्वोन्मत्तं विद्यात्; असकृत्स्व- प्नरोदनहास्यं नृत्यगीतवाद्यपाठकथान्नपानस्नानमाल्यधूपगन्धरति रक्तविप्लुताक्षं द्विजातिवैद्यपरिवादिनं रहस्यभाषिणं च यक्षोन्मत्तं विद्यात्; नष्टनिद्रमन्नपानद्वेषिणमनाहारमप्यतिबलिनं शस्त्र-शोणितमांसरक्तमाल्याभिलाषिणं संतर्जकं च राक्षसोन्मत्तं विद्यात्; प्रहासनृत्यप्रधानं देवविप्रवैद्यद्वेषावज्ञाभिः स्तुतिवेदमन्त्र–

शास्त्रोदाहरणैः काष्ठादिभिरात्मपीडनेन च ब्रह्मराक्षसोन्मत्तं विद्यात्; अस्वस्थचित्तं स्थानमलभमानं नृत्यगीतहासिनं बद्धाबद्धप्रलापिनं संकरकूटमलिनरथ्याचेलतृणा- श्मकाष्ठाधिरोहणरतिं भिन्नरूक्षस्वरं नग्नं विधावन्तं नैकत्र तिष्ठन्तं दुःखान्यावेदयन्तं नष्टस्मृतिं च पिशाचोन्मत्तं विद्यात् ॥ *C.S.Ci. 9/20*

देवोन्मत्त उन्माद (Devonmatta Unmāda)

Characteristic features of insanity caused by gods are as follows : Calm look, serious, invincible, unwrathful, no desire for sleep and food, having small quantity of sweat, urine, stool and flatus, emitting auspicious odour and having the face like blossomed lotus flower.

गुरु वृद्धादि शापजन्य उन्माद (Guru Vṛddhādi Śāpajanya Unmāda)

One having the activities, diet and speech corresponding to curse, magical spell, desire to transform on the basis of will power, elders, accomplished persons & sages is to be diagnosed as suffering from *Guru Vṛddhādi Śāpajanya Unmāda.*

पितृभिरुन्मत्त उन्माद (Pitṛbhirunmatta Unmāda)

Characteristic features of *Unmāda* caused by the forefathers are as follows:– Unhappy look, no vision, excessive sleep, interrupted speech, loss of desire for food, anorexia and indigestion.

गन्धर्वोन्मत्त उन्माद (Gandharvonmatta Unmāda)

Characteristic features of *Unmāda* caused by the seizure of *Gandharva* are as follows : Voilent acts, excess bravery, sharpness, seriousness, invincibility and fondness for musical instruments played by mouth, dancing, singing, food, drink, bath, garland, incense, perfumes, red apparel, offerings, funnytales, questioning and emits auspicious smell from the body.

यक्षोन्मत्त उन्माद (Yakṣonmatta Unmāda)

Characteristic features of *Unmāda* caused by the seizure of *Yakṣa* are as follows : Frequent sleep; weeping, laughing; fondness for dance, vocal & instrumental music, recitation of sacred scriptures, talking stories, food, drink, garlands, incense & perfumes; eyes are red & agitated; despises *Brāhmaṇās* & physicians and discloses secrets of others.

राक्षसोन्मत्त उन्माद (Rakṣasonmatta Unmāda)

Characteristic features of *Unmāda* caused by the seizures of demons are as follows : Insomnia, aversion to food & drink, excessive strength inspite of eating small amount of food or fasting, liking for weapons, blood, meat, & red garlands and one who is ferocious.

ब्रह्मराक्षसोन्मत्त उन्माद (Brahma Rākṣasonmatta Umnāda)

Characteristic features of *Unmāda* caused by the seizure of a class of evil demons are as follows : Excessive loud laughing, dancing; express aversion & disobedience to gods, *Brāhmaṇās* & physicians, quotes illustrations from humans, the *Vedās*, and other scriptures and self infliction by pieces of wood etc.

पिशाचोन्मत्त उन्माद (Piśāconmatta Unmāda)

Characteristic features of *Unmāda* caused by the seizure of a particular sections of demons are as follows : Fickle mindedness, does not find peace anywhere, engages himself in dancing, singing, laughing, & incoherent speech, fond of climbing on garbage, dirty lanes, cloth, grasses, stones, wood, voice is broken & hoarse, remains naked & runs here & there, complains of his sorrows before others and suffers from loss of memory.

Time period for the attack of various exogenous seizures

तत्र चौक्षाचारं तपःस्वाध्यायकोविदं नरं प्रायः शुक्लप्रतिपदि त्रयोदश्यां च छिद्रमवेक्ष्याभिधर्षयन्ति देवाः; स्नानुशुचिविविक्तसेविनं धर्मशास्त्रश्रुतिवाक्यकुशलं प्रायः षष्ठ्यां नवम्यां चर्षयः; मातृपितृगुरुवृद्धसिद्धाचार्योपसेविनं प्रायो दशम्याममावस्यायां च पितरः; गन्धर्वाः स्तुतिगीतवादित्ररतिं परदारगन्धमाल्यप्रियं चौक्षाचारं प्रायो द्वादश्यां चतुर्दश्यां च, सत्वबलरुपगर्वशौर्ययुक्तं माल्यानुलेपनहास्यप्रिय मति वाक्प्रवणं प्रायःशुक्लैकादश्यां च यक्षाः; स्वाध्यायतपोनियमोपवासब्रह्मचर्यदेवयतिगुरु- पूजाऽरतिं भ्रष्टशौचं ब्राह्मण णवादिनं शूरमानिनं देवागारसलिलक्रीडनरतिं प्रायः शुक्लपञ्चभ्यां पूर्णचन्द्रदर्शने च ब्रह्माणमब्राह्मणं वा

ब्राह्मणवादिनं शूरमानिनं देवागारसलिलक्रीडनरतिं प्रायः शुक्ल प्ञ्चभ्यां पूर्णचन्द्रदर्शने च ब्रह्मराक्षसाः रक्षःपिशाचास्तु हीनसत्त्वं पिशुनं स्तेनं लुब्धं शठं प्रायो द्वितीयातृतीयाष्टमीषु; इत्यपरिसंख्येयानां ग्रहाणामाविष्कृततमा ह्यष्टावेते व्याख्याताः ॥ *C.S.Ci. 9/21*

Devonmatta Unmada

God attacks the person with pure conduct & engaged in austerity and study by finding loopholes after on the first or thirteenth day of the bright fortnight.

Guru Vṛddhādi Śāpajanya Unmāda

Sages posses the persons fond of bath, purity and lonely place, and conversant with the saying of the religious scriptures and the *Vedās* generally on the sixth or ninth day of the fortnight in an opportunate moment.

Pitṛbhirunmatta Unmāda

Fore feathers enter into the person who is engaged in the service of mother partner, teacher, elders, accomplished persons and preceptors often on tenth day of the dark fortnight or new moon.

Gandharvonmatta Unmāda

Gandharvās enter into the person who is fond of praising verses, vocal & instrumental music, women of other persons, perfumes, garlands, purity & good conduct often on twelfth and fourteenth day of the fortnight.

Yakṣonmatta Unmāda

Yakṣās possess the persons endowed with mental & physical strength, good complexion, ego & valour, having liking for garlands, unction & laughter and who is talkative generally during the seventh or eleventh day of the bright fortnight.

Brahmarākṣasonmatta unmāda

Brahmarākṣasās enter into the person who has dislike for study, austerity, religious practices, fasting, celibacy, and worship of gods, ascetics, & preceptor; lost interest in cleanliness, whether *Brāhmaṇa* or not says himself as *Brāhmaṇa,* regards himself as brave, has fondness for playing in temples & water tanks, often on the fifth day of the bright fortnight or full moon.

Rakṣasonmatta and Piśāconmatta Unmāda

Rakṣasās & Piśācās attack the person who has inferior psyche, who is a back biter, thief, greedy & wicked often on the second, third, or eighth day of the fortnight.

साध्यासाध्यता (Sādhyāsādhyatā)

सर्वेष्वपि तु खल्वेषु यो हस्तावुद्यम्य रोषसंरम्भान्निःशङ्कमन्येष्वात्मनि वा निपातयेत् स ह्यसाध्यो ज्ञेयः तथा यः साश्रुनेत्रो मेढ्रप्रवृत्तरक्तः क्षतजिह्वः प्रस्रुतनासिकश्छिद्यमानचर्माऽप्रतिहन्यमानपाणिः सततं विकूजन् दुर्वर्णस्तृषार्तः पूतिगन्धश्च स हिंसार्थिनोन्मत्तो ज्ञयः; तं परिवर्जयेत् ॥ *C.S.Ci. 9/22*

Of all the varieties of *Unmāda,* if the patient strikes at others or himself without hesitation raising his hands with fit, anger and agitation should be considered as incurable.

One who has tearful eyes, hemorrhage from penis, wounded tongue, running nose, abraised skin; uninterrupted speech, constant mumbling, deranged complexion, excessive thirst, putrid smell from the body indicates person is suffering from *Unmāda* as a result of possession of violent spirits and should be rejected for treatment.

मानस्ज उन्माद (Mānasaja Unmāda)

चौरैर्नरेन्द्रपुरुषैररिभिस्तथाऽन्यैर्वित्रासितस्य धनबान्धवसङ्क्षयाद्वा । गाढ़ं क्षते मनसि च प्रियया रिरंसोर्जायेत चोत्कटतरो मनसो विकारः ॥ *S.S.Utt. 62/12*

Persons getting threatened by thieves, officers of the king (robbers, murderers etc.), enemies & such others : or by loss of wealth and relatives; or when greatly desirous of copulating with the beloved causing injuries to the mind leading to manifestation of *Unmāda*. These patients talks differently, reveals the desires of his mind without knowing, sings, laughs or weeps.

मदज उन्माद (Madaja Unmāda)

चित्रं स जल्पति मनोऽनुगतं विसंज्ञो गायत्यथो हसति रोदिति मूढसंज्ञः ॥ *S.S.Utt. 62/13*

It is characterised by reddish eye, loss of strength, sense perception & complexion; timidness, bluish face and looks like gioing to die soon.

उपशय/पथ्य (Upaśaya/Pathya)

Helpful measures and diets (pathyas) in cases of Unmāda

In cases of the *Unmāda* caused by the disordered *Doṣas*, the patient first of all should be administered oil therapy, vomitting (*Vamana*), *Virecana* (purgation), *Basti* therapy, *Nasya*, blood letting, fomentation and the like. One should also cause actions to make the patient fear, to get him consoled and delighted. He should be beaten, bathed, ointmented, allowed to sleep and smoke. The following edibles are helpful: wheat, *Mudga* beans, red variety of *Śāli* rice, nature-warm milk, *Śatadhauta Ghṛtam*, fresh as well as old *Ghṛtam*, meat of the tortoise and wild animals and birds, mango fruit, old ash-gourd, *Paṭola*, *Brāhmī*, *Vāstuka* leaves, *Tanḍulīya* leaves, urines of ass and horse, rain water, *Haraḍa*, *Svarṇa Bhasma*, coconut fruit, grapes and raisin, jackfruit and *Kapittha*. (B.R. 24/102-106)

Pathyas in cases of Unmāda caused by passion and grief

The patient of such cases should be offered provisions contrary to the situation disturbing him. Optionally, the insanity caused by sexual passion should be removed by non-desire, the grief related cases should be removed by delighting the patient. The cases of fear-generated insanity could be cured by brave thoughts, anger-generated cases by peace, greed-generated cases by thoughts of donations, and the likewise. If the insanity is caused by loss of some item, the desired items could be provided to the patient. (B.R. 24/107-108)

Useful measures in cases of Unmāda caused by celestial elements

If the insanity has been caused due to the anger of gods, seers, deceased ancestors, *Gandharvās* and the like, administration of strong and harsh majors such as *Nasya* (nasal treatments), strong collyrium, and beating or the like must be avoided. The following measures are useful: worshipping the celestial agency concerned, offering sacrifices and presentations, performing the holy fire rituals (*Havana*), chanting of the holy *Gāyatṛī Mantras*, keeping religious fasts, following the right kind of social conducts and behaviours including being truthful in speech and deeds, performing holy duties and repentations, wearing purposeful and holy gems and jewelry, worshipping God *Śiva* and the holy figures like *Guhyaka* and *Pramāḍhaka* wandering on earth as well as the priests. (B.R. 24/109-111)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Avoidable activities during the cases of Unmāda : Taking liquor, incompatible foods, taking hot meals, excessive sleeping, suppression of natural urges and hunger and thirst, having intercourse, taking fruits of the *Āṣāḍha* month (rainy seasons), eating *Kārvellaka* and leafy vegetables, bitter foods including *Kathillaka*, and the like. (B.R. 24/112-113)

अपस्मार (Apasmāra)

Introduction

Epilepsy; falling sickness; a violent fit or seizure; fits with loss of memory. *Apasmāra* is a disease which comes in paroxysms and due to the derangement of *buddhi* (intellect) and the *manas* (mind), the patient loses his memory or consciousness for a brief period. It comes in paroxysm; patient sees imaginary things and falls down; his eyes and eyebrows become distorted; there is a discharge of foaming from his mouth; he feels tremors; his hand and feet become convulsed; the paroxysm lasts for a brief period and the patient regains his consciousness as if he is awaking from a sleep.

अपस्मार निरुक्ति (Apasmāra Nirukti)

स्मृतिर्भूतार्थविज्ञानमपश्च परिवर्जने ।
अपस्मार इति प्रोक्तस्ततोऽयं व्याधिरन्तकृत् ॥
S.S.Utt. 61/3

One who lost the experience of the past and knowledge of nature of things is known as *Apasmāra* & it is going to kill the person.

स्मृतेरपगमं प्राहुरपस्मारं भिषग्विदः ।
तमःप्रवेशं बीभत्सचेष्टं धीसत्त्वसंप्लवात् ॥
C.S.Ci. 10/3

Apasmāra is defined by experts as departure of memory associated with entering into darkness & loathsome appearance due to derangement of intellect and mind.

अपस्मारं पुनः स्मृतिबुद्धिसत्वसंप्लवाद्बीभत्सचेष्टमावस्थिकं तमः प्रवेशमाचक्षते॥ *C.S.Ni. 8/5*

Apasmāra is defined as the transient appearance of consciousness with loathsome expression due to derangement of memory, intelligence and mind.

अपस्मार हेतु (Apasmāra Hetu)

विभ्रान्तबहुदोषाणामहिताशुचिभोजनात् ।
रजस्तमोभ्यां विहते सत्त्वे दोषावृते हृदि ॥
चिन्ताकामभयक्रोधशोकोद्वेगादिभिस्तथा ।
मनस्यभिहते नृणामपस्मारः प्रवर्तते ॥
C.S.Ci. 10/4-5

मिथ्याऽतियोगेन्द्रियार्थकर्मणामभिसेवनात् ।
विरुद्धमलिनाहारविहारकुपितैर्मलैः ॥
वेगनिग्रहशीलानामहिताशुचिभोजिनाम् ।
रजस्तमोऽभिभूतानां गच्छताञ्च रजस्वलाम् ॥
तथा कामभयोद्वेगक्रोधशोकादिभिर्भृशम् ।
चेतस्यभिहते पुंसामपस्मारोऽभिजायते ॥
S.S.Utt. 61/4-6

Etiological factors for the manifestation of *Apasmāra* are as follows : Consumption of unwholesome & unclean food, infliction of mind with *Rajas* and *Tamas,* occlusion of the *Hṛdaya* by the excacerbated *Doṣās*, affliction to mind by excessive anxiety, passion, fear, anger, grief, agitation etc.; improper and excess correlation of the sense organs with their objects and of the activities; incompatible & contaminated foods, improper activities, suppression of the natural urges and sexual intercourse during menstruation. All the above etiological factors causes the perversion of mind due to excessive accumulation of *Doṣās* leading to development of *Apasmāra*.

त एवंविधानां प्राणभृतां क्षिप्रमभिनिर्वर्तन्ते; तद्यथा-रजस्तमोभ्यामुपहतचेतसामुद्भ्रान्तविषमबहुदोषाणां समलविकृतोपहितान्यशुचीन्यव्यवहारजातानि वैषम्ययुक्तेनोपयोगोविधि नोपयुञ्जानानां तन्त्रप्रयोगमपि च विषममाचरतामन्याश्च शरीरचेष्टा विषमाः समाचरतामत्युपक्षयाद्वा दोषाः प्रकुपित्ता रजस्तमोभ्यामुपहतचेतसामन्तरात्मनः श्रेष्ठतममायतनं हृदयमुपसृत्योपरि तिष्ठन्ते, तथेन्द्रियायतनानि च। तत्र चावस्थिताः सन्तो यदा हृदयमिन्द्रियायतनानि चेरिताः कामक्रोधभयलोभमोहहर्षशोकचिन्तोद्वेगादिभिः सहसाऽभिपूरयन्ति, तदा जन्तुरपस्मरति॥ *C.S.Ni. 8/4*

Epilepsy manifest in those individuals whose mind is disturbed by *Rajas & Tamas;* who have scattered, abnormal & plenty of *Doṣās,* one who doesn't follow application of rules & regulation of diet, *Tāntrik* practices improperly, abnormal body postures or due to wasting *Doṣās* get vitiated and, in those having mind disturbed by *Rajas & Tamas,* spread over to *Hṛdaya,* the best seat of inner self, and also the seats of sense organs and stay there predominantly while residing there when they are excited by passion, anger, fear, greed, confusion, exhilaration, grief, anxiety, agitation etc. & fill up the *Hṛdaya* & seats of sense organ as a result person gets bouts of *Apasmāra*.

अपस्मार संप्राप्ति एवं लक्षण (Apasmāra Samprāpti evam Lakṣaṇa)

संज्ञावहेषु स्त्रोतःसु दोषव्याप्तेषु मानवः ।
रजस्तमःपरीतेषु मूढो भ्रान्तेन चेतसा ॥
विक्षिपन् हस्तपादं च विजिह्मभ्रूविलोचनः ।
दन्तान् खादन् वमन् फेनं विवृताक्षः पतेत् क्षितौ ॥
अल्पकालान्तरञ्चापि पुनः संज्ञां लभेत सः ।
सोऽपस्मार इति प्रोक्तः॥
S.S.Utt. 61/8-10

धमनीभिः श्रिता दोषा हृदयं पीडयन्ति हि ।
संपीड्यमानो व्यथते मूढो भ्रान्तेन चेतसा ॥
C.S.Ci. 10/6

Due to etiological factors *Doṣās* (both *Śārīrika* and *Mānasika*) become excessively excacerbated and reaches *Samjñavaha Srotas* via *Dhamani* and manifest *Apasmāra.* The person thus affected, is afflicted with stupor & malafunctioning of mental activities due to *Rajas & Tamas,* makes violent movement of the arms, legs, brows, and eyes;

grinding of teeth, froath comes out from mouth, he sees non-existent things, excessive salivation, falls on the ground with eyes open and regain consciousness after a bouts of convulsant attacks as if from sleep. These are the characteristic features of *Apasmāra.*

Nidāna Sevana (Consumption of Etiological factors)

↓

Aggravation of *Doṣās* both *Śārīrika* and *Mānasika (Rajas & Tamas,)*

↓

reaches *Samjñavaha Srotas* via *Dhamani*

↓

Get localised in the *Samjñavaha Srotas*

↓

Amalgamation of *doṣās* & *dūṣyās* in the localized area

↓

Apasmāra

अपस्मार संप्राप्ति घटक (Samprāpti Ghaṭaka of Apasmāra)

Doṣa	Both *Śārīrika* and *Mānasika (Raja & Tamas)* *Vāta → Vyāna, Prāṇa, Udāna* *Pitta → Sādhaka*
Dūṣya	*Dhātu → Rasa*
	Upadhātu → Dhamani *Śārīrika Mala → (Raja & Tamas)* *Dhātu Mala → Sveda, Kapha*
Agni	*Jaṭharāgni*
Agniduṣṭi	*Mandatā*
Āma	*Āgnijanya*
Srotas	*Samjñavaha Srotas, Rasavaha Srotas*
Srotoduṣṭi	*Saṅga, Vimārgagamana*
Udbhavasthāna	*Hṛdaya*
Adhiṣṭhāna	*Manas, Indriya*
Vyaktasthāna	*Mana & Śarīra*
Sancārasthāna	*Hṛdayagata Dhamani*
Svabhāva	*Āśukāri, Cirakāri*
Prabhāva	*Ekadoṣaja Sādhya* *Tridoṣaja Asādhya*
Rogabheda	4 Types
Roga Mārga	*Madhyama*

अपस्मार पूर्वरूप (Apasmāra Pūrvarupa)

तस्येमानि पूर्वरूपाणि भवन्ति; तद्यथा- भ्रूव्युदासः सततमक्ष्णोर्वैकृत मशब्दश्रवणं लालसिङ्घाणप्रस्त्रवणमनन्नाभिषण-मरोचकाविपाकौ हृदयग्रहः कुक्षेराटोपो दौर्बल्यमस्थिभेदोऽङ्गमर्दो मोहस्तमसो दर्शनं मूर्च्छा भ्रमश्चाभीक्ष्णं स्वप्ने च मदनर्तनव्यध नव्यथनवेपनपतनादीनीति। *C.S.Ni. 8/6*

हत्कम्पः शून्यता स्वेदो ध्यानं मूर्च्छा प्रमूढता।
निद्रानाशश्च तस्मिंस्तु भविष्यति भवन्त्यथ॥
S.S.Utt. 61/7

Premonitory signs & symptoms of *Apasmāra* are as follows : Contraction of eye brows, frequent abnormal movement of eyes, hearing of such sounds as are nonexistent, excessive discharge of saliva & nasal excreta, disinclination for food, anorexia, indigestion, stiffness in cardiac region, distension of abdomen, debility, tearing pain in bones, bodyache, unconsciousness, feeling of darkness infront of eyes, fainting, giddiness, dreams of narcosis, dancing, piercing, aching, shivering, falling etc., tremors & feeling of emptiness of the heart, sweating, worry, & loss of sleep.

अपस्मार रोग भेद (Apasmāra Roga Bheda)

स च दृष्टश्चतुर्विधः।
वातपित्तकफैर्नृणाञ्चतुर्थः सन्निपाततः॥
S.S.Utt. 61/10-11

इह खलु चत्वारोऽपस्मारा भवन्ति वातपित्तकफसन्निपात-निमित्ताः। *C.S.Ni. 8/3*

Apasmāra is classified into 4 varieties namely *1. Vātaja, 2. Pittaja, 3. Kaphaja* and *4. Sannipātaja.*

वातज अपस्मार (Vātaja Apasmāra)

वेपमानो दशन् दन्तान् श्वसन् फेनं वमन्नपि।
यो ब्रूयाद्विकृतं सत्त्वं कृष्णं मामनुधावति।
ततो मे चित्तनाशः स्यात्सोऽपस्मारोऽनिलात्मकः॥
S.S.Utt. 61/11-12

तत्रेदमपस्मारविशेषविज्ञानं भवति; तद्यथा- अभीक्ष्णमपस्मरन्तं, क्षणेन संज्ञां प्रतिलभमानम्, उत्पिण्डिताक्षम्, असाम्ना विलपन्तम्,

उद्वमन्तं फेनम्, अतीवाध्मातग्रीवम्, आविद्धशिरस्कं, विषमविनताङ्गुलिम्, अनवस्थितपाणिपादम्, अरुणपरुषश्यावनखनयनवदनत्वचम्, अनवस्थितचपलपरुषरूक्षरूपदर्शिनं, वातलानुपशयं, विपरीतोपशयं च वातेनापस्मरन्तं विद्यात्॥ *C.S.Ni. 8/8*

Characteristic features of *Vātaja Apasmāra* are as follows: Frequent fits followed by regaining consciousness instantaneously, protruded eyes, crying recklessly, emitting froath from the oral cavity, excessive heaviness & rigidity of neck, bending of the head to one side, irregularly contracted fingers, instability of hands & feet, reddish, dry, blackish nails, eyes, face & skin, vision of unstable, fickle, coarse & dry objects; aggravation of signs & symptoms by *Vāta* aggravating factors and get relieved by *Vāta* alleviating regimens. Tremors, bites his teeth, dyspnoea, patient says that abnormal & black substances are running after him and one who lost his *Citta* (mind).

पित्तज अपस्मार (Pittaja Apasmāra)

अभीक्ष्णमपस्मरन्तं, क्षणेन संज्ञां प्रतिलभमानम्, उत्पिण्डिताक्षम्, असाम्ना विलपन्तम्, उद्वमन्तं फेनम्, अतीवाध्मातग्रीवम्, आविद्धशिरस्कं, विषमविनताङ्गुलिम्, अनवस्थितपाणिपादम्, अरुणपरुषश्यावनखनयनवदनत्वचम्, अनवस्थितचपलपरुषरूक्षरूपदर्शिनं, वातलानुपशयं, विपरीतोपशयं च वातेनापस्मरन्तं विद्यात्॥ *C.S.Ni. 8/8*

तृट्तापस्वेदमूर्च्छार्त्तो धुन्वन्नङ्गानि विह्वलः।
यो ब्रूयाद्विकृतं सत्त्वं पीतं मामनुधावति।
ततो मे चित्तनाशः स्यात्स पित्तभव उच्यते॥
S.S.Utt. 61/13-14

Characteristic features of *Pittaja Apasmāra* are as follows : Frequent fits followed by regaining consciousness instantaneously, breathing with sounds, rubbing earth; green, yellow and coppery nails, eyes, face & skin; vision of bloody, agitated, fierce, luminous & irritated objects, signs & symptoms aggravate by *Pitta* provocative measures & subside by *Pitta* alleviating factors. Patient also suffers from thirst, warmness, sweating, fainting, shaking of body parts, one who is agitated, patient says that abnormal yellow substances are running after him and lastly patients looses his *Citta* (mind).

कफज अपस्मार (Kaphaja Apasmāra)

चिरादपस्मरन्तं, चिराच्च संज्ञां प्रतिलभमानं पतन्तम्, अनतिविकृतचेष्टं, लालामुद्वमन्तं, शुक्लनखनयनवदनत्वचं, शुक्लगुरुस्निग्धरूपदर्शिनं, श्लेष्मलानुपशयं, विपरीतोपशयं च श्लेष्मणाऽपस्मरन्तं विद्यात्॥ *C.S.Ni. 8/8*

शीतहृल्लासनिद्रार्त्तः पतन् भूमौ वमन् कफम्॥
यो ब्रूयाद्विकृतं सत्त्वं शुक्लं मामनुधावति।
ततो मे चित्तनाशः स्यात्सोऽपस्मारः कफात्मकः॥
S.S.Utt. 61/14-15

Characteristic features of *Kaphaja Apasmāra* are as follows : Delayed fits followed by delayed recovery, falling down, absence of much distortion activities, dribbling of saliva, white nails, eyes, face & skin, vision of white, heavy and unctous objects, signs & symptoms aggravate by *Kapha* provocative measures & get relieved by *Kapha* pacifying regimen. Patient also suffers from cold, nausea, excessive sleep, vomits *Kapha*, patient says that abnormal white substances is running after him and lastly patient looses his *Citta* (mind).

सन्निपातज अपस्मार (Sannipātaja Apasmāra)

समवेतसर्वलिङ्गमपस्मारं सान्निपातिकं विद्यात्, तमसाध्यमाचक्षते॥ *C.S.Ni. 8/8*

Characteristic features of *Sannipātaja Apasmāra* are as follows : Appearance of all the signs & symptoms of all varieties of *Doṣaja Apasmāra* and which is incurable.

साध्यासाध्यता (Sādhyāsādhyatā)

अपस्मारः स चासाध्यो यः क्षीणस्यानवश्च यः॥
पक्षाद्वा द्वादशाहाद्वा मासाद्वा कुपिता मलाः।
अपस्माराय कुर्वन्ति वेगं किंचिदथान्तरम्॥
C.S.Ci. 10/12-13

Prognosis of *Apasmāra* are as follows : *Apasmāra* if manifest in emaciated persons and which persist for longer period of time is said to be incurable. The aggravated *Doṣās* cause attacks of *Apasmāra* once in fifteen days, twelve days, or a month. The attack may, however, takes place even after a shorter period.

Whether *Apasmāra* is *Doṣajany* or not?

Apasmāra manifest without any apparent cause, because of disappearing without any treatment and because of scriptures describing it differently, some scholars predict that it is not produced by the *Doṣās*. But *Suśruta* says that *Doṣās* become aggravated gradually, because of their instability (*Doṣās* attain various stage quickly) because of the description in all the scriptures (mentions the disease causation by the *Doṣās*) and because of having many kinds, intelligent persons definitely say that the disease is born from *Doṣās* only. Just as the seed in the ground sprouts only during *Śarad ṛtu*, though it rains in other seasons, similarly this disease develops from the *Doṣās* which are staying in the body getting aggravated in very short time & manifesting its different symptoms by their nature itself. Hence the disease *Apasmāra* is dreadful & manifest by abnormal *Doṣās*. (S.S.Utt. 61/17-21)

उपशय/पथ्य (Upaśaya/Pathya)

The wise physicians have suggested that all the *Nasya* (nasal) therapy, *Añjana* (collyrium) therapy, and drugs prescribed in the case of *Unmāda* can be also used for treatment of *Apasmāra* as well. (B.R. 20/54)

The helpful measures for the cases of *Apasmāra* include the following: nasal therapy, *Śirā Vedhana* (cutting of the veins), giving donations, beating, use of fear, use of force, smoke therapy, demonstration of amazing scenes, consolation, bathing, ointment of oily substances, and the like. The helpful meals for the patients of epilepsy include the red variety of *Śāli* rice, *Mudga* beans, wheat, flesh of tortoise, juice of wild animals and birds, milk, *Brāhmī Patra*, *Vacā*, *Paṭola Patra*, ash gourd, *Vāstuka* leaves, sweet pomegranate, *Śobhāñjana* (drum-sticks), coconut, grapes, *Āmalakī*, *Parusaka*, sesame oils, urines of ass and horse, rain water and *Harītakī*. (B.R. 25/55-59)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The harmful measures and meals for the cases of *Apasmāra* include the following: grief, brooding, fear, anger, taking unclean eatables, liquor, fishes, unwholesome and incompatible foods, very hot, sour and heavy to digest foods, excessive intercourse and exercising, violating the honour of the deceased elderly, gods and other respectable agencies, all kinds of vegetable leaves, *Bimbī* leaves, rainy season fruits, holding the urge of sleep and thirst, sitting inside water, mounting on trees and hills, and the like. (B.R. 25/60-62).

Unmada & Apasmara

MCQs

1. *Asphalayanthambhumi* is the feature of
   a. *Vatajaapasmara*
   b. *Kaphajaapasmara*
   c. *Pittajaapasmara*
   d. *Tridoshajaapasmara*
2. *Souchadwesha* is the feature of
   a. *Kaphajaunmada* b. *Vatajaunmada*
   c. *Agantujaunmada* d. *Graha*
3. *Sangyavaha srotodusti* observed in
   a. *Apasmara* b. *Unmada*
   c. *Attwabhinivesha* d. *Graha*
4. The disease *Unmada* manifest due to vitiation of which *srotas*?
   a. *Manovaha Srotas*
   b. *Sagyavaha Srotas*
   c. *Buddhivahasira*
   d. *Indriyavaha Srotas*
5. *Utpinditaksha* is the feature of
   a. *Vatajaunmada* b. *Vatajaapasmara*
   c. *Pittajaunmada* d. *Pittajaapasmara*
6. *Rahaskamata* is the feature of
   a. *Kaphajaapasmara* b. *Agantujaunmada*
   c. *Kaphajaunmada* d. *Vatajaunmada*
7. Types of *Unmada* as per *Susruta*
   a. 4 b. 5
   c. 6 d. 3
8. *Aniyataschaunmadakala* is the feature of
   a. *Vatajaapasmara* b. *Pittajaunmada*
   c. *Kaphajaunmada* d. *Agantujaunmada*
9. The affliction by Devagrahaunmada is by
   a. *Avalokana* b. *Sapa*
   c. *Sparsa* d. *Samavesa*

10. *Visesharupa* of *Vatajaapasmara* mentioned by *Susruta*

a. *Sula* b. *Hrittoda*
c. *Jrimbha* d. *Utkleda*

Answer

1.	c	2.	a	3.	a
4.	a	5.	a	6.	c
7.	c	8.	d	9.	a
10.	b				

CHAPTER
51

Vishada

Prof. Parameswarappa S. Byadgi and Dr. Anurag Pandey

Etymology and Definitions

असिद्धि भयात् विविधेषु कर्मेषु अप्रवृत्ति विषादः ।
विषादो अनुष्येतो आत्मनं अशक्तजननं।
विषादः सर्वदा मनः क्लेदः।
अवसादः सदा जाढ्यं मौर्ख्यं। (श.क.द्रु.)

1. *Viṣāda* is derived from the words *vi*, *ṣāda* and *kta* according to *Śabdakalpadrūmam*
2. It is the combination of vi and *ṣāda*; *dhajpratyaya* according to *Vācaspatyam*
3. *Vācaspatyam*

अवसादः यथाजाट्यं.........विषदोअवसादाः सादोविषादः ।
(भगवद्गीता)

The terms *avaṣāda* and *jāṭya* are mentioned as synonyms of *viṣāda* (*Vācaspatyam*).

4. Monier Williams gives the meaning of *viṣāda* as drooping state, lassitude, depression and languor
5. *Viṣāda* is witnessed in the *Arjuna Viṣāda Yoga*, the first chapter of *Bhagavad Gīta*, where *Arjuna* mighty warrior describes his helplessness condition as :

सिद्धन्तिममगात्रणिमुखचपरिशुष्यति, वेपथुचशरीररोमहषेवजायते, गाण्टीवंस्त्रंसतेहस्ततादत्वक्चएवं परिदह्यते ।

i.e. *Arjuna* says, my limbs feel weak and loose, my mouth dried up with heat, tremors all over my body and I feel horripilation, my bow slips from my hand and my skin is burning all over, my brain is whirling and I can't stand any longer.

विषादश्च, अतिप्रलापश्च, रौक्ष्यं च, पारुष्यं च, श्यावारुणावभासता च, अस्वप्नश्च, अनवस्थितचित्तत्वं च; इत्यशीतिर्वातविकारा वातविकाराणामपरिसंख्येयानामाविष्कृततमा व्याख्याताः ॥ *C.S.Su 20/11*

Viṣāda is a *vātajanānātmaja vikāra.*

विषादो रोगवर्धनानां। *C.S.Su 25.40*

It has also been addressed to be *Agrya* for *Rogavardhanām.*

एचच्चैव कारणमपेक्षमाणा हीनबलमातुरमविषादकरैर्मृदुसुकुमारप्रायैरुत्तरोत्तरगुरुभिरविभ्रमैरनात्ययिकैश्चोपचरन्त्यौषधैः विशेषतश्चनारीः। *C.S.Vi. 8/54*

भयं विषादेन, धैर्यमविषादेन । *C.S.Vi. 4.8*

One can perceive a person's fear by observing his depression.

Free from depression shows his courage.

यथा वातक्षये उत्साहविरोधिनो विषादस्य वृद्।
C.S. Su 18/52

Cakrapāṇi comments *vātakṣaya* increases *viṣāda.*

असिद्धिभयाद्विविधेषु कर्मसु सादोऽप्रवृत्तिः विषादः ।
S.S.Su. 1/25 (Dalhaṇa)

Dalhaṇa explains, i.e. a condition originated from apprehension of failure, results in incapability of mind and body to function properly with significant reduction in activity.

शूरो विषादरहितः। *S.S.Su 34/20 (Dalhaṇa)*

Dalhaṇa also mentions braveness as the characteristics seen in the absence of depression.

वाक्कायचितावसादो विषादः। *S.S.K. 3/21 (Dalhaṇa)*

Viṣāda is the state of *avaṣāda* which is characterised by lassitude of spirit, body and mind, where the physical and mental retardation of the individual is understood.

12. *Aṣṭāṅga Hṛdaya* says as a state of dejection or diminished interest.

15. Also as mainly caused by *tamadoṣa* as well as it occurs in *hīnasatva* person on seeing blood and flesh of animals or man.

16. *Satva sāra* person is not affected by *viṣāda.*

17. *Cakrapāṇi* comments in the context states that *viṣāda* as *lakṣaṇa* of *vātājajvara.*

18. *Satva* will be seen altered in case of *viṣāda.*

Aetiology

The classical *nidānās* (*Caraka*) explained for *mānasikarogās* can be considered as basic *nidāna* of *viṣāda- Asātmendriyārthasamyoga*, *Prajnāparādhā*, *Pariṇāma*.

Ahara (Diet)

- Irregularity in taking food and frequently skipping meals
- Eating at late night, junk foods, salty, fermented foods regularly in excess quantity etc.
- *Kaṭurasa- bhrama, mada, mūrchā, dāha, santāpa*
- *Tiktarasa - stambha, bhrama, mada*
- Improper food habits like *samaśana*, *adhyaśana*, *viṣamāśana* can cause *viṣāda* because of *prajnāparādhā*.
- Loss of self control over sense organs with respect to taking food and excessive greediness for various food items.

Vihāra

Ativyavāya

शुक्रं धैर्यम् च्यवन देहवलं हर्षे भोजार्थ च । *S.Su.15/4(2)*

Ativyavāyā (excessive sexual intercourse) may cause *viṣāda*

Avyāyāma

Avyāyāma- lack of physical and mental exercise found to be major causative factor in the pathogenesis of depression

Mānasika

Dukha, bhaya, irṣyā, dainya, lobha, cintā, krodha causes *viṣāda*.

Rogajanya

Secondary to diseases like *Vātaja jvara, Viṣa* and all the somatic disease after attaining chronicity leads to depression.

Prajnapāradhā

Improper activities pertaining to body, mind and speech cause *viṣāda* like bad lifestyle habits such as overwork, lack of sleep, poor diet and lack of regular exercise are also responsible.

Asātmyendriyārtha Samyoga

- Unwholesome contact with the objects may be in the form of *atiyoga, ayoga* and *mithyā yoga.*
- It crosses the threshold range of sense organs, stressful condition will cause imbalance of *Doṣa.*
- Also some use and withdrawal of addictives like alcohol, caffeine, nicotine etc.

Pariṇāma

- *Maturity* of *kāla* and *karma*
- Results of all misdeeds mature by time
- Seasonal affective disorder (SAD) - mood disorder
- Transylvanian effect- effect of moon on human biology.
- Power of full moon to cause disorders of mind, insanity and even epilepsy.

Pathogenesis

- The *samprāpti* can be designed using available references and on the basis of *doṣa*, *dūṣya* etc. involved.

हीनसत्त्वास्तु नात्मना नापि परैः सत्त्वबलं प्रति शक्यन्ते उपस्तम्भयितुं, महाशरीरा ह्यपि ते स्वल्पानामपि वेदनानामसहा दृश्यन्ते, सन्निहितभयशोकलोभमोहमाना रौद्रभैरवद्विष्टबीभत्सविकृतसंकथास्वपि च पशुपुरुषमांसशोणितानि चावेय विषादवैवर्ण्यमूर्च्छोन्मादभ्रमप्रपतनानामन्यतममाप्नुवन्त्यथवा मरणमिति ॥ *C.S Vi 8.119*

1. Inferior type of mental faculties.
2. Neither by themselves nor through others can sustain their mental strength.
3. Inspite of having sound physique, they cannot susceptible to fear, grief, greed, delusion and ego.
4. When they hear even stories describing wrathful, fearful, hateful, terrifying and ugly situation or come across visions of flesh or blood of an animal or man, they fall victims to depression, pallor, fainting, madness, giddiness of falling on the ground, or such events may even lead them to death.

5. These types of persons are contraindicated to *saṁśodhana karma.*
6. Tolerates and adjusts to pain or odd situations very badly.
7. Predominant *tamoguṇa* individuals cannot tolerate at all.

If *hinasatva/hinasara* person is exposed to psychological stress like fear, loss and failure in life, grief, worry, envy, disliking etc. causes the vitiation of *manodoṣās* (rajo, tamo) associated with exacerbated vata, kaphadosha along with *āvaraṇa* of tamas leading to abnormality in the *manovaha srotas* and produce *Viṣāda.*

Samprāpti Ghataka

Doṣa : *Mānasika doṣās- tama and raja Sāriradoṣās- prāṇa, udāna and vyānavāta, sadhaka pitta and tarpakakapha* are involved.
Dhātu : *Rasādi dhātu*
Agni : *Jaṭharāgnimāndya*
Srotas : *Manovaha, Rasavaha*
Srotoduṣṭi : *Saṅga*
Udbhavasthāna : *Hṛdaya*
Vyaktasthāna : *Sarva Śarīra*
Adhiṣṭhāna : *Manovahasrotas , Hṛdaya*
Sancārasthāna : *Manovahasrotas*
Vyakthastāna : *Manas and Śarīra*
Prabhāva : *Kṛcchrasādhya*
Svabhāva : *Āśukāri*

Symptoms

In Suśruta Saṁhit?,

1. *Viṣāda* is explained by *Dalhaṇa* as *Asiddhi-* Unable to get the desired object
2. *Bhaya-* Fear from known and unknown objects
3. *Sadānd Apravṛtti-* No interest and non-indulgence in work.
4. According to the references in *Bhagavad Gīta,* the somatic symptoms like.
   - *Sidanti Gātrāṇi* (loosening of muscles)
   - *Mukhaśoṣa* (dry mouth)
   - *Vepathu* (tremors)
   - *Aruci* (anorexia)
   - *Prasveda* (sweating)
   - *Tvak Paridāha* (burning sensation in the skin)
   - *Prasveda* (excessive sveda)
   - *Romaharṣa* (horripilation)
   - *Sramṣanam* (inability to hold)

विपादकरंशरीरमानसीःग्लानिकरं ।

Indicates a dejected or coward state of mind and body. The states of *dhi* the function of decision making and *dhṛti-* the power of will are impaired.

Mental symptoms like

- *Dūkhītā* (distressed, unhappy, grieved in mind)
- *Avaṣāda* (hopelessness, lassitude)
- *Manah khedah* (feeling of depression)
- *Viśanna* (dejection, sorrow)
- *Citta glāni* (fatigue of mind)
- *Asiddhi bhaya* (fear and anxiety or apprehension of failure)
- *Dainya* (miserable state)
- *Bhramatimanas* (wandering mind)

MCQs

1. *Avasada* and *jadhya* are the synonyms of

   a. *Vishada* b. *Bhaya*
   c. *Atatvabhinivesa* d. *Mada*

2.असिद्धिभयात्विविधेषुकर्मेषुअप्रवृत्ति________

   a. *Kaphajaapasmara*
   b. *Madatyaya*
   c. *Vishada*
   d. *Kaphajaunmada*

3. *Vishada* included in

   a. *Vatajananatmajavikara*
   b. *Pittajananatmajavikara*
   c. *Kaphajananatmajavikara*
   d. *Sarvadoshakopa*

4. As per *Caraka* which among the following cause *Vishada*

   a. *Kaphavridhi* b. *Kaphakshaya*
   c. *Vatakshaya* d. *Pitta kshaya*

5. *Agrya* for *Rogavardhana*
   a. *Virudhahara*
   b. *Vishado*
   c. *Agnidushti*
   d. *Ekarasa asana*

Answer

1.	a	2.	c	3.	a
4.	c	5.	b		

CHAPTER 52

Murchha and Sanyasa

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मूर्च्छा (Mūrcchā)

Introduction

Mūrcchā is an indisposition characterised by the transient loss of consciousness. In this case, the patient may become conscious after sometime even without a treatment. Synocope, Loss of senses. When, in a person because of emaciation, excessive accumulation of *doṣa*, indulgence in mutually contradictory food, suppression of manifested natural urges, injury and weakness of mind, aggravated *doṣa* enter into the seats of external and internal *Indriyās* (motor and sensory organs) then he faints. When the channels carrying sensation (*Sajñāvaha nāḍī*) get obstructed by *vāyu* etc. darkness appears all of a sudden and the afflicted person becomes incapable of experiencing favourable and unfavourable stimulations as a result of which he falls like a log of wood. This condition is called *moha* (unconsciousness) and *mūrcchā* (fainting). This is of six categories.

There are six varieties of *Mūrcchā*. They are called *Vātika mūrchā*, *Paittika mūrchā*, *Kaphaja mūrchā*, *Sannipātika mūrchā*, *Raktaja mūrchā*, *Viṣaja* and *Madhyaja mūrcchā* depending on the causes involved. In all these varieties, *pitta* plays an important role. Pain in cardiac region, yawning, *glāni* (drowsiness and feeling of heaviness in the cardiac region) and loss of consciousness-these are the premonitory signs and symptoms of *mūrcchā* (fainting). Thereafter, the specific variety of the disease is manifested.

मूर्च्छा हेतु (Mūrcchā Hetu)

क्षीणस्य बहुदोषस्य विरुद्धाहारसेविनः।
वेगाघातादभीघाताद्धीनसत्त्वस्य वा नः॥
करणायतनेषूग्रा बाह्येष्वाभ्यन्तरेषु च।
निविशन्ते यदा दोषास्तदा मूर्च्छन्ति मानवाः॥

S.S.Utt. 46/3-4

Persons who are emaciated, in whom the *Doṣās* greatly aggravated, one who consume incompatible foods, suppression of natural urges, who are injured, & those who are mentally inferior.

मूर्च्छा संप्राप्ति (Mūrcchā Samprāpti)

Due to above etiological factors *Doṣās* become greatly excacerbated and accumulate in both the external & internal sense organs resulting into the development of *Mūrchā.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Vātādi Doṣās*
↓
Doṣās accumulate in both the external & internal sense organs
↓
Amalgamation of *Doṣās* & *Dūṣyās* in the external & internal rogas
↓
Mūrchā

मूर्च्छा पूर्वरूप (Mūrcchā Pūrvarūpa)

हृत्पीडा जृम्भणं ग्लानिः संज्ञानाशो बलस्य च।
सर्वासां पूर्वरूपाणि, यथास्वं ता विभावयेत्॥

S.S.Utt. 46/8

Premonitory signs & symptoms of *Mūrchā* are pain in the heart, yawning, exhaustion, loss of consciousness & strength.

प्रत्यात्म लक्षण (Pratyātma Lakṣaṇa)

संज्ञावहासु नाडीषु पिहितास्वनिलादिभिः।
तमोऽभ्युपैति सहसा सुखदुःखव्यपोहकृत्॥
सुखदुःखव्यपोहाच्च नरः पतति काष्ठवत्।
मोहो मूर्च्छेति तां प्राहुः॥

S.S.Utt. 46/5-6

Due to obstruction in the *Sajñāvaha Srotas* by the increased *Vātādi Doṣās*, then Tamas (darkness) enhances greatly & destroys the feeling of the happiness & misery; by the destruction of feeling of comfort & discomfort, the person falls on the ground like a log of wood is known as *Moha* or *Mūrchā*.

षड्विधा सा प्रकीर्त्तिता ॥
वातादिभिः शोणितेन मद्येन च विषेण च ।
षट्स्वप्येतासु पित्तं हि प्रभुत्वेनावतिष्ठते ॥

S.S.Utt. 46/6-8

It is of six varieties i.e.

i) *Vātaja*, ii) *Pittaja*, iii) *Kaphaja*, iv) *Raktaja* v) *Madhyaja* vi) *Viṣaja*. Among all types *Pitta* is the predominant *Doṣa*

वातज मूर्च्छा (Vātaja Murchā)

नीलं वा यदि वा कृष्णमाकाशमथवाऽरुणम् ।
पश्यंस्तमः प्रविशति शीघ्रं च प्रतिबुध्यते ॥
वेपथुश्चाङ्गमर्दश्च प्रपीडा हृदयस्य च ।
कार्श्यं श्यावाऽरुणाच्छाया मूर्च्छाये वातसंभवे ॥

M.Ni. 17/7-8

Vātaja Mūrchā patients sees the sky in blue, black or crimson colours, goes into darkness, falls down unconscious but regains consciousness quickly, tremors, general malaise, severe pain in the heart, emaciation and blackish red discolouration of the body.

पित्तज मूर्च्छा (Pittaja Mūrcchā)

रक्तं हरितवर्णं वा वियत्पीतमथापि वा ।
पश्यंस्तमः प्रविशति सस्वेदश्च प्रबुध्यते ॥
संपिपासः ससन्तापो रक्तपीताकुलेक्षणः ।
जातमात्रे पतति च शीघ्रं च प्रतिबुध्यते ।
संभिन्नवर्चाः पीताभो मूर्च्छाये पित्तसंभवे ॥

M.Ni. 17/9-10

Pittaja Mūrcchā patients sees sky appearing as red, green or yellow colours, loss of consciousness and regaining it after perspiration, (Thirst, increased body temperature, eyes red or yellow, loss of consciousness & quick regaining) diarrhoea & yellowish discolouration of the body.

कफज मूर्च्छा (Kaphaja Mūrcchā)

मेघसंकाशमाकाशमावृतं वा तमोघनैः ।
पश्यंस्तमः प्रविशति चिराच्च प्रतिबुध्यते ॥
गुरुभिः प्रावृतैरङ्गैर्यथैवार्द्रेण चर्मणा ।
सप्रसेकः सहृल्लासो मूर्च्छाये कफसंभवे ॥

M.Ni. 17/11-12

Kaphaja Mūrcchā patients sees the sky full of thick clouds, falls unconscious, regains consciousness after long time, feels as though his body is covered with heavy moist leather, salivation & nausea.

सन्निपातज मूर्च्छा (Sannipātaja Mūrcchā)

सर्वाकृतिः सन्निपातादपस्मार इवागतः ।
स जन्तुं पातयत्याशु विना बीभत्सचेष्टितैः ॥

M.Ni. 17/13

Sannipātaja Mūrcchā patients develops symptoms of all the three *Doṣās*, symptomatology mimic *Apasmāra* but without *Bibhatsaceṣṭā*.

रक्तज मूर्च्छा (Raktaja Mūrcchā)

पृथिव्यम्भस्तमोरूपं रक्तगन्धश्च तन्मयः ।
तस्माद्रक्तस्य गन्धेन मूर्च्छन्ति भुवि मानवाः ।
द्रव्यस्वभाव इत्येके दृष्ट्वा यदभिमुह्यति ॥

S.S.Utt. 46/10

Rakta is composed of *Pṛthvi* & *Āpa Bhūtās* which have predominant *Tamoguṇa*. Some patients become unconscious & fall to the ground after seeing & smelling the blood. Some people attribute it as the specific property of blood.

रक्तज मूर्च्छा (Raktaja Mūrcchā)/विषज मूर्च्छा (Viṣaja Mūrcchā)/मद्यज मूर्च्छा (Madhyaja Mūrcchā)

गुणास्तीव्रतरत्वेन स्थितास्तु विषमद्ययोः ॥
त एव तस्मात्ताभ्यां तु मोहौ स्यातां यथेरितौ ।
सत्ब्धाङ्गदृष्टिसत्वसृजा गूढोच्छ्वासश्च मूर्च्छितः ॥
मद्येन विलपञ्शेते नष्टविभ्रान्तमानसः ।
गात्राणि विक्षिपन् भूमौ जरां यावन्न याति तत् ॥

M.Ni. 7/15-17

Stiffness of the body & fixed gaze are observed in *Raktaja Mūrcchā*.

Inconsistent speech, falling on the ground with loss of mental activities, abnormal movements of the body, these symptoms disappear soon after the digestion of alcohol are the characteristic features of *Madhyaja Mūrcchā*. *Madhyaja Mūrcchā* is caused by poisonous & alcohol drinks having profound symptoms. Tremors, sleep, thirst & feeling of darkness & other symptoms specific to the particular poisonous substances are the characteristic features of *Viṣaja Mūrcchā*.

Predominance of *Doṣās* in *Mūrcchā*, *Bhrama*, *Tandra* & *Nidrā*

मूर्च्छा पित्ततमःप्राया रजःपित्तानिलाद् भ्रमः ।
तमोवातकफात्तन्द्रा निद्रा श्लेष्मतमोभवा ॥
M.Ni. 17/19

Mūrcchā is caused by predominance of *Pitta* & *Tamas; Bhrama* is caused by *Rajas, Pitta* & *Vāta*. *Tandrā* manifest due to predominance of *Tamas, Vāta* & *Kapha*. *Nidrā* is caused by predominance of *Kapha* & *Tamas*.

भ्रम रोग (Bhrama Roga)

चक्रवद् भ्रमतो गात्रं भूमौ पतति सर्वदा ।
भ्रमरोग इति ज्ञेयो रजःपित्तानिलात्मकः ॥
M.Ni. 17/19

Bhrama Roga is characterised by reeling of a body like a turning wheel & person falls on the ground regularly. It manifest due to predominance of *Raja, Pitta* & *Vāta*.

तन्द्रा (Tandrā)

इन्द्रयार्थेष्वसंवित्तिर्गौरवं जृम्भणं क्लमः ।
निद्रार्तस्येव यस्येहा तस्य तन्द्रां विनिर्दिशेत् ॥
M.Ni. 17/20

Tandrā is characterised by non perception of their objects by the sense organs, heaviness, yawning, feeling exhaust without performing any physical activities & person appears like sleeping.

सन्यास (Sannyāsa)

प्रभूतदोषस्तमसोऽतिरेकात्सम्मूर्च्छितो नैव विबुध्यते यः ।
संन्यस्तसंज्ञो भृशदुश्चिकित्स्यो ज्ञेयस्तदा बुद्धिमता मनुष्यः ॥
S.S.Utt. 46/21

Sannyāsa manifest due to excacerbation of *Doṣās* or of predominance of *Tamoguṇa* and it is characterised by non awakening state or unconsciousness. It is difficult for treatment.

दोषेषु मदमूर्च्छायाः कृतवेगेषु देहिनाम् ।
स्वयमेवोपशाम्यन्ति, संन्यासो नौषधैर्विना ॥
वाग्देहमनसां चेष्टामाक्षिप्यातिबला मलाः ।
संन्यासं सन्निपतिताः प्राणायतनसंश्रयाः ॥
कुर्वन्ति, तेन पुरुषः काष्ठीभूतो मृतोपमः ।
म्रियेत शीघ्रं शीघ्रं चेच्चिकित्सा न प्रयुज्यते ॥
अगाधे ग्राहबहुले सलिलौघ इवात्तटे ।
संन्यासे विनिमज्जन्तं नरमाशु निवर्तयेत् ॥
A.H.Ni. 6/36-39

Patient regain consciousness soon after bouts of attacks in case of *Mūrcchā* & *Mada*. However in case of *Sannyāsa* patient does not regain consciousness without treatment.

Excessively excacerbated powerful *Doṣās* invade the seats of *Prāṇāyatana* and brings about impairment of the speech, body & mind. Patients appears like a log of wood & a dead man. If it is not managed immediately patient dies soon.

Patients of *Sannyāsa* is sinking in the ocean of *Sannyāsa* which is very deep infested with many crocodiles, having huge waves & no shore should be abandoned soon.

उपशय/पथ्य (Upaśaya/Pathya)

The following activities are advisable for a patient suffering from the disease of *Mūrcchā* (syncope): smoking (herbal ingredients), applying *Añjana* to eye lids, nasal treatment, cauterization, prickling with needles, pulling the hair, causing pain beneath his nail plates, cutting him by teeth, closing the passage of his nose and mouth so that breathing is checked, allowing him to vomit, to fast, frightening him or showing anger to him, putting him on a bed upon which he could not sleep properly, telling him pleasing stories, placing him under cool shadows, rain water, applying the *Ghṛtam* over his forehead, etc. The patient should take hot syrup prepared by the fresh rice-grains, old barley, the *Śāli* variety of red rice-grains, the old *Ghṛtam* (kept in some earthen pitcher), the syrup of the peas and *Mudga* (green gram), juice of meat of the animal and birds from the *Jāṅgala* regions, the pickles and confectioneries, cow milk, sugar candy, old ash pumpkin, leaves of *Paṭola*, banana, *Harītakī*, grains that are easily digestible, water of wells located by the side of rivers, white sandalwood and water treated with camphor. The sands of the river land, sounds, show of strange items, singing loudly and making large instrumental sounds before the patients, all these are helpful items in cases of syncope. One is also advised to remind the patient of the lost family members, of the lost memories, his self-knowledge and to make the patient gain consolation, etc. (B.R. 21/22-27)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The items to be a avoided while suffering from *Mūrcchā* (syncope) includes the eatables like the betel leaves, leaves of mustard saplings, buttermilk, etc. and such acts as using the tooth-sticks to clean the tooth, facing the sun, taking incompatible meals, having intercourse, sweating, taking bitter items, holding the urge of thirst and sleep. (B.R. 21/28)

MCQs

1. आहारपरिणामान्तेभूयश्चलभतेबलम is clinical feature of
   a. व्यपेताहिक्का (Vyapeta Hikk
1. *Kalahapriyata* is the feature of
   a. *Paithikamada* b. *Paithikamurcha*
   c. *Dwitiyamada* d. *Dhvamsaka*
2. *Apasmara iva patatyasu nischeshtam vinabhebhtsa ceshtitaihi* is the feature of
   a. *Vishajamada* b. *Tridoshajamurcha*
   c. *Sanyasa* d. *Tritiyamada*
3. *Kashtibhoothomritopamama* is the feature of
   a. *Tritiyamada* b. *Vikshaya*
   c. *Sanyasa* d. *Madyajamada*
4. Number of *Madatyaya upadrava* as per *Madavanidana*
   a. 4 b. 7
   c. 10 d. 11
5. *Pretasahabhakshanam* in *Svapna* is seen in
   a. *Jwara* b. *Udara*
   c. *Apasmaram* d. *Madatyaya*
6. Which among the following is not the *Guna* of *Madhya*
   a. *Amla* b. *Katu*
   c. *Ruksha* d. *Sukshma*
7. *Nischeshtashavavat shete is the feature of*
   a. *Tridoshajamurcha*
   b. *Tridoshaja Madatyaya*
   c. *3rd stage of mada*
   d. *Sanyasa*
8. *Srotas* involved in the *sampraphti* of *Mada, Murcha & Sanyasa*
   a. *Mamsa, Meda & Satwa*
   b. *Asthi, Majja & Chetana*
   c. *Shukra, Majja & Manas*
   d. *Rasa, Raktha & Chetana*
9. *Meghasankasam pasyanaakasham* is the feature of which *murcha*
   a. *Vata* b. *Pitta*
   c. *Kapha* d. *Tridoshaja*
10. *Dosha* predominance of Sanyasa
    a. *Vata- Kaphaja* b. *Vata - Pittaja*
    c. *Tridoshaja* d. *Kapha - Pittaja*
11. *Agade graha bahula salilougha ivaathare* is told about
    a. *Mada* b. *Madatyaya*
    c. *Murcha* d. *Sanyasa*

Answer

1.	a	2.	b	3.	c
4.	b	5.	d	6.	b
7.	c	8.	d	9.	c
10.	c	11.		d	

CHAPTER

53

Common Psychiatric Diseases

Prof. Parameswarappa S. Byadgi and Dr. Anurag Pandey

Depression

Psychiatric disorders

1. These are characterised by the emotion of anxiety, worrisome thoughts, avoidance behaviour and the somatic symptoms of autonomic arousal.
2. The nature and prominence of the somatic symptoms often lead the patient to present initially to medical services.
3. Anxiety may be stress related and phobic anxiety may follow an unpleasant incident.
4. Patients with anxiety often also have depression.
5. Anxiety disorders are divided into three main subtypes: phobic, paroxysmal (panic) and generalised

Classification of anxiety disorders

	Phobic anxiety disorder	Panic disorder	Generalised anxiety disorder
Occurrence	Situational	Paroxysmal	Persistent
Behaviour	Avoidance	Escape	Agitation
Cognitions	Fear of situation	Fear of symptoms	Worry
Symptoms	On exposure	Episodic	Persistent

Phobic anxiety disorder

1. A phobia is an abnormal or excessive fear of an object or situation, which leads to avoidance of it (such as excessive fear of dying in an air crash leading to avoidance of flying).
2. A generalised phobia of going out alone or being in crowded places is called agoraphobia.
3. Phobic responses can develop to medical interventions such as venepuncture.

Panic disorder

1. Panic disorder describes repeated attacks of severe anxiety, which are not restricted to any particular situation or circumstances.
2. Somatic symptoms such as chest pain, palpitations and paraesthesiae in lips and fingers are common.
3. The symptoms are in part due to involuntary over-breathing (hyperventilation).
4. Patients often fear they are suffering from a serious illness such as a heart attack or stroke, and may therefore seek emergency medical attention.
5. Panic disorder is often associated with agoraphobia.

Generalised anxiety disorder

1. This is chronic anxiety associated with uncontrollable worry.
2. Somatic symptoms of muscle tension and bowel disturbance often lead to a medical presentation.

Obsessive-compulsive disorder

1. Obsessive-compulsive disorder (OCD) is characterised by obsessive thoughts, which are recurrent, unwanted and usually anxiety provoking, and by compulsions, which are repeated acts performed to relieve feelings of tension.
2. An example is repeated hand-washing because of thoughts of contamination.
3. The differential diagnosis is from normal checking behaviour and from delusional beliefs about thought possession.
4. OCD is equally common in men and women.

Mood or effective disorders

Mood or affective disorders include:

- *Unipolar depression:* one or more episodes of low mood and associated symptoms
- *Bipolar disorder:* episodes of elevated mood interspersed with episodes of depression

- *dysthymia:* chronic low-grade depressed mood without sufficient other symptoms to count as clinically significant or major depression.

Depression

1. Major depressive disorder has a prevalence of 5% in the general population and approximately 10% in chronically ill medical out patients.
2. It is a major cause of disability and of suicide.
3. If comorbid with a medical condition, depression magnifies disability, diminishes adherence to medical treatment and rehabilitation, and may even shorten life expectancy.
4. Such comorbid depression may incrementally worsen health more than any combination of chronic diseases without depression.

Aetiology

1. There is a genetic predisposition to depression, especially when of early onset.
2. The number and identity of the genes are largely unknown but the serotonin transporter gene is a candidate.
3. Adversity and emotional deprivation early in life also predispose to depression.
4. Depressive episodes are often triggered by stressful life events (especially those that involve loss), including medical illnesses.
5. Associated biological factors include hypofunction of monoamine neurotransmitter systems (5-HT and noradrenaline (norepinephrine)) and abnormal hypothalamo-pituitary-adrenal axis (HPA) regulation, which results in elevated cortisol levels that do not suppress with dexamethasone.

Diagnosis

1. Depression may be mild, moderate or severe, and episodic, recurrent or chronic.
2. Pointers to an organic cause for psychiatric disorder
    - Late age of onset of psychiatric illness
    - No previous history of psychiatric illness
    - No family history of psychiatric illness
    - No apparent psychological precipitant

Depressed Mood

Presenting Problems in Psychiatric Illness

Anxiety symptoms–

1. Anxiety may be transient, persistent, episodic or limited to specific situations.
2. Most commonly it is transient, as an adjustment disorder and subsides with time.
3. Anxiety may occasionally be a manifestation of a medical condition such as thyrotoxicosis

Symptoms of anxiety disorder

Psychological

Apprehension
Irritability
Worry
Fear of impending disaster
Poor concentration
Depersonalisation

Somatic

Palpitations
Fatigue
Tremor
Dizziness
Sweating
Diarrhoea
Frequent desire to pass urine
Chest pain
Initial insomnia
Breathlessness
Headache

Differential diagnosis of anxiety

- Normal response to threat
- Adjustment disorder
- Generalised anxiety disorder
- Panic disorder
- Phobic disorder
- Organic (medical) cause
- Hyperthyroidism
- Paroxysmal arrhythmias
- Pheochromocytoma
- Alcohol and benzodiazepine withdrawal
- Hypocalcaemia
- Temporal lobe epilepsy

Depressed mood

1. Depressive disorder is common, occurring in a quarter to a half of medical in patients.
2. The symptoms of depression are both mental and physical.

3. In the physically ill patient, diagnosis of comorbid depression is based mainly on careful assessment of the core psychological symptoms: namely, persistently lowered mood and anhedonia.

Symptoms of depressive disorders

Psychological

- Depressed mood
- Loss of interest
- Reduced self-esteem
- Loss of enjoyment (anhedonia)
- Pessimism
- Guilt
- Suicidal thinking

Somatic

- Reduced appetite
- Loss of libido
- Weight change
- Bowel disturbance
- Disturbed sleep
- Motor retardation (slowing of activity)
- Fatigue

Differential diagnosis

1. Depressive disorder must be differentiated from an adjustment disorder with depressed mood
2. Adjustment disorders are common, self-limiting reactions to adversity, including physical illness, which are transient and require only general support.
3. On the other hand, depressive disorders are characterised by more severe and persistent mood disturbance and do require specific treatment.
4. In some cases, depression may occur as a result of a direct effect of a medical condition or its treatment on neurotransmitters or neural pathways in the brain, when it is referred to as an 'organic mood disorder.

Organic affective disorders

Neurological Infections :

- Cerebrovascular disease
- Infectious mononucleosis
- Cerebral tumour
- Herpes simplex
- Multiple sclerosis
- Brucellosis
- Parkinson's disease
- Typhoid
- Huntington's disease
- Toxoplasmosis
- Alzheimer's disease

Connective tissue disease

- Epilepsy
- Systemic lupus erytematosus

Endocrine	Drugs
• Hypothyroidism	• Reserpine, methyldopa
• Hyperthyroidism	• Phenothiazines
• Cushing's syndrome	• Phenylbutazone
• Addison's disease	• Corticosteroids, oral contraceptives
• Hyperparathyroidism	
Malignant disease	• Interferon

Suicide

1. Depression is the major risk factor for suicide, especially when it co-occurs with a medical condition or substance misuse.
2. When depression is suspected, tactful enquiry should always be made into suicidal thoughts and plans.
3. Asking about suicide does not increase the risk of it occurring, whereas failure to enquire denies the opportunity to prevent it.

Risk factors for suicide

- Psychiatric illness (depressive illness, schizophrenia)
- Older age
- Male sex
- Living alone
- Unemployed
- Recently bereaved, divorced or separated
- Chronic physical ill health
- Drug or alcohol misuse
- Suicide note written

- History of previous attempts (especially if a violent method was used)

Elated mood

1. Elation, or euphoria, is the converse of depression and is characteristic of mania.
2. It may manifest as infectious joviality, over-activity, lack of sleep and appetite, undue optimism, over-talkativeness, irritability, and recklessness in spending and sexual behaviour.
3. When more severe, psychotic symptoms are often evident, such as delusional degrees of grandiosity.
4. Elevated mood is much less common than depression, and where it arises in medical settings is often secondary to drug or alcohol misuse, an organic disorder or medical treatment.

Delusions and hallucinations

Delusions

1. Various types of delusion are identified on the basis of their content.
2. They may be:
   - Persecutory, such as a conviction that others are out to get one
   - Hypochondriacal, such as an unfounded conviction that one has cancer
   - Grandiose, such as a belief that one has special powers or status.
3. The most extreme form of delusion is nihilistic, e.g. 'My head is missing; 'I have no body; 'I am dead.' Delusions should be differentiated from overvalued ideas, which are strongly held but not fixed.

Hallucinations

1. These are perceptions without external stimuli. They can occur in any sensory modality, most commonly visual or auditory.
2. Typical examples are hearing voices when no one else is present, or seeing 'visions'.
3. Hallucinations have the quality of ordinary perceptions and are perceived as originating in the external world, not the patient's own mind (when they are termed pseudo-hallucinations).
4. Those occurring when falling asleep ('hypnagogic') and on waking ('hypnopompic') are not pathological.
5. Hallucinations should be distinguished from illusions, which are misperceptions of real external stimuli (such as mistaking a shrub for a person in poor light).

Anxiety neurosis

Introduction and Management

Anxiety is a normal reaction to stress and can actually be beneficial in some situations. For some people, however, anxiety can become excessive. Anxiety disorders are a group of mental disorders characterized by feelings of anxiety and fear. Anxiety is a worry about forthcoming events and fear is a response to present events. The term anxiety covers four aspects of experiences that the person may have; mental uneasiness, physical pressure, physical symptoms and dissociative anxiety. These feelings may cause physical symptoms, such as a palpitation and trembling.

The emotions present in anxiety disorders range from simple nervousness to bouts of terror. Other psychiatric and medical problems may mimic with the symptoms of an anxiety disorder such as hyperthyroidism. Anxiety disorders occur about twice as often in females as males, and generally begin during childhood.

Classification

There are several forms of anxiety disorders. According to Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision, also known as DSM-IV-TR, a manual published by the American Psychiatric Association (APA) that includes all currently recognized mental health disorders, the following is the classification of Anxiety disorders.

1. Generalized anxiety disorder
2. Panic disorder with agoraphobia
3. Panic disorder without agoraphobia
4. Agoraphobia without history of panic disorder
5. Specific phobia

6. Social phobia
7. Obsessive-compulsive disorder
8. Posttraumatic stress disorder
9. Acute stress disorder
10. Anxiety disorder due to a general medical condition
11. Anxiety disorder NOS

Apart from the above, there are some other conditions, which are clinically found. They are Separation Anxiety, Situational Anxiety.

Generalized Anxiety Disorder (Gad)

Generalized anxiety disorder is the most common anxiety disorder to affect adults. It is a common chronic disorder characterized by long-lasting anxiety that is not concentrated on any one object or situation. Those suffering from generalized anxiety disorder experience non-specific persistent fear and worry, and become overly concerned with everyday matters.

Generalized anxiety disorder is "characterized by chronic excessive worry accompanied by three or more of the following symptoms: restlessness, fatigue, concentration problems, irritability, muscle tension, and sleep disturbance". Anxiety can be a symptom of a medical or substance abuse problem, and medical professionals must be aware of this.

Clinical presentation : The patient looks anxious, with increased sweating from the hands, feet, and axillae,and he/she may be weeping, which can suggest depression. Before a diagnosis of anxiety disorder is made, the physician must rule out drug-induced anxiety and other medical causes. In children GAD may be associated with headaches, restlessness, abdominal pain, and heart palpitations. Typically, it begins around 8 to 9 years of age.

Diagnosis: A diagnosis of GAD is made when an individual has been disproportionately anxious about an everyday problem for six months or more. A person may find that he/she has problems making daily decisions and remembering commitments as a result of lack of concentration/ preoccupation with worry.

Panic Disorder: In panic disorder, an individual suffers from brief attacks of extreme terror and anxiety, often marked by trembling, shaking, confusion, dizziness, nausea, and/or difficulty breathing. These panic attacks, defined by the APA as fear or discomfort that abruptly arises and peaks in less than ten minutes, can last for several hours. Attacks can be triggered by stress, fear, or even exercise; the specific cause is not always apparent.

Clinical presentation

As such, those suffering from panic disorder experience symptoms even outside specific panic episodes. Often, normal changes in heartbeat are noticed by a panic sufferer, leading them to think something is wrong with their heart or they are about to have another panic attack. In some cases, a heightened awareness (hypervigilance) of body functioning occurs during panic attacks, wherein any perceived physiological change is interpreted as a possible life-threatening illness.

Diagnosis : A diagnosis of panic disorder requires that said attacks have chronic consequences: either worry over the attacks' potential implications, persistent fear of future attacks, or significant changes in behavior related to the attacks.

Agoraphobia

Agoraphobia is the specific anxiety about being in a place or situation where escape is difficult or uncomfortable or where help may be unavailable. Agoraphobia is strongly linked with panic disorder and is often precipitated by the fear of having a panic attack.

Clinical presentation : A common expression involves needing to be in constant view of a door or other escape route. In addition to the fears themselves, the term agoraphobia is often used to refer to avoidance behaviors that sufferers often develop. For example, following a panic attack while driving, someone suffering from agoraphobia may develop anxiety over driving and will therefore avoid driving. These avoidance behaviors can often have serious consequences.

Phobias: Phobic disorders are the single largest category of anxiety disorders. It includes all cases in which fear and anxiety is triggered by a definite stimulus or situation. Between 5% and 12% of the population worldwide, suffer from phobic disorders. Sufferers typically anticipate terrifying consequences from encountering the object of their fear, which can be anything from an animal to a location to a bodily fluid to a particular situation. Sufferers understand that their fear is not proportional to the actual potential danger but still are overwhelmed by the fear.

Social Anxiety Disorder : Social anxiety disorder (SAD; also known as social phobia) defines an intense fear and avoidance of negative public scrutiny, public embarrassment, humiliation, or social interaction. This anxiety can be specific to particular social situations for e.g. as public speaking. More classically, it is experienced in most or all-social interactions. Social anxiety often manifests specific physical symptoms, including blushing, sweating, and difficulty in speaking. As with all phobic disorders, those suffering from social anxiety often will try to escape from the source of their anxiety. However, in the case of social anxiety this is particularly problematic. In severe cases can lead to complete social isolation.

Obsessive Compulsive Disorder

Obsessive–compulsive disorder (OCD) is a type of anxiety disorder primarily characterized by repetitive obsessions which are distressing, persistent, and intrusive thoughts or images and compulsions such as urges to perform specific acts or rituals. It affects roughly 3% of the population worldwide.

The OCD thought pattern may be likened to superstitions. Often the process is entirely illogical. In addition, in many cases, the compulsion is entirely inexplicable, simply an urge to complete a ritual triggered by nervousness.

Post-Traumatic Stress Disorder

Post-traumatic stress disorder (PTSD) is an anxiety disorder that results from a traumatic experience. Post-traumatic stress can result from an extreme situation, such as combat, natural disaster, rape, hostage situations, child abuse, bullying, or even a serious accident. It can also result from long-term (chronic) exposure to a severe stressor. Common symptoms include hyper vigilance, flashbacks, avoidant behaviors, anxiety, anger and depression. There are a number of treatments that form the basis of the care plan for those who are suffered with PTSD. Such treatments include cognitive behavioral therapy (CBT), psychotherapy and support from family and friends.

Acute Stress Disorder

Acute stress disorder is characterized by the development of severe anxiety, dissociative, and other symptoms that occurs within one month after exposure to an extreme traumatic stressor, e.g. witnessing a death or serious accident. As a response to the traumatic event, the individual develops dissociative symptoms. Individuals with Acute Stress Disorder have a decrease in emotional responsiveness, often finding it difficult or impossible to experience pleasure in previously enjoyable activities, and frequently feel guilty about pursuing usual life tasks.

A person with acute stress disorder may experience difficulty concentrating, feel detached from their bodies, experience the world as unreal or dreamlike, or have increasing difficulty recalling specific details of the traumatic event.

Anxiety Disorder Due to a General Medical Condition

In this, anxiety disorder due to a general medical condition, the individual experiences anxiety that causes clinically significant distress or impairment in functioning. This anxiety must represent a direct physiological, not emotional, consequence of a general medical condition.

Symptoms can include prominent, generalized anxiety symptoms, Panic Attacks, or obsessions or compulsions. There must be evidence from the history, physical examination, or laboratory findings that the disturbance is the direct physiological consequence of a general medical condition. The disturbance is not better accounted for mental disorder, such as adjustment disorder with Anxiety, in which the stressor is the general medical condition. The diagnosis is not made if the anxiety symptoms occur only during the course of a delirium. The anxiety symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

In determining whether the anxiety symptoms are due to a general medical condition, the clinician must first establish the presence of a general medical condition. Further, the clinician must establish that the anxiety symptoms are etiologically related to the general medical condition through a physiological mechanism. A careful and comprehensive assessment of multiple factors is necessary to make this judgment. Although there are no infallible guidelines for determining whether the relationship between the anxiety symptoms and the general medical condition is etiological, several considerations provide some guidance in this area. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of the general medical condition and the anxiety symptoms. A second consideration is the presence of features that are atypical of a primary anxiety disorder (e.g., atypical age at onset or course, or absence of family history). Evidence from the literature that suggests that there can be a direct association between the general medical condition in question and the development of anxiety symptoms may provide a useful context in the assessment of a particular situation.

Anxiety Disorder not Otherwise Specified

This group comprises the disorders with prominent anxiety or phobic avoidance and that they do not meet criteria for any specific Anxiety Disorder, Adjustment Disorder with Anxiety, or Adjustment Disorder with Mixed Anxiety and Depressed Mood. The following are the examples of this group.

1. Mixed anxiety-depressive disorder: clinically significant symptoms of anxiety and depression, but the criteria are not met for either a specific Mood Disorder or a specific anxiety disorder

2. Clinically significant social phobic symptoms that are related to the social impact of having a general medical condition or mental disorder e.g., Parkinson's disease, dermatological conditions, Stuttering, Anorexia Nervosa, Body Dysmorphic Disorder)

3. Situations in which the disturbance is severe enough to warrant a diagnosis of an anxiety disorder but the individual fails to report enough symptoms for the full criteria for any specific anxiety disorder to have been met. The examples are an individual who reports all of the features of Panic Disorder without Agoraphobia except that the panic attacks are all limited-symptom attacks.

4. Situations in which the clinician has concluded that an anxiety disorder is present but is unable to determine, whether it is primary, or due to a general medical condition, or substance induced.

Separation Anxiety

Separation anxiety disorder (SepAD) is the feeling of excessive and inappropriate levels of anxiety over being separated from a person or place. Separation anxiety is a normal part of development in babies or children, and it is only when this feeling is excessive or inappropriate that it can be considered a disorder. Separation anxiety disorder affects roughly 7% of adults and 4% of children, but the childhood cases tend to be more severe; in some instances, even a brief separation can produce panic.

Situational Anxiety

Situational anxiety is caused by new situations or changing events. It can also be caused by various events that make that particular individual uncomfortable. Its occurrence is very common. Often, an individual will experience panic attacks or extreme anxiety in specific situations. A situation that causes one individual to experience anxiety may not affect another individual, at all. For example, some people become uneasy in crowds, tight spaces, so standing in a tightly packed line, say at the bank, or a store register, may cause them to experience extreme anxiety, possibly a panic attack. Others, however, may experience anxiety when major changes in life occur, such as entering college, getting married, having children, etc.

Anxiety Disorders In Children

Children may experience anxiety disorders similar to adults.

A common anxiety disorder in children is school phobia, which in some cases can be a type of separation anxiety. Sometimes the anxiety has no obvious cause. In other instances, the child may experience maltreatment from classmates, or even a teacher. They could also be stressed from the workload they are given. School phobia may also be a form of social phobia, also known as social anxiety. Children with this disorder may avoid speaking in front of their classmates or meeting new people. Typically, social phobia in children it is caused by some traumatic event, such as not knowing an answer when called on in class.

Aetiology of Anxiety Disorders

1. Drugs: alcohol abuse; Caffeine, alcohol and benzodiazepine dependence; exposure to organic solvents in the work environment;
2. Stress: Life stresses such as financial worries or chronic physical illness;
3. Genetics: GAD runs in families and is six times more common in the children of someone with the condition.
4. Persistence of anxiety:
5. Evolutionary mismatch

Epilepsy

1. A seizure is any clinical events caused by an abnormal electrical discharge in the brain, whilst epilepsy is the tendency in have recurrent seizures.
2. Epilepsy should be regarded as a symptom of brain disease rather than a disease itself.
3. A single seizure is not epilepsy but an indication for investigation, and medication should generally be with held until recurrent seizures occur.
4. The recurrence rate after a first seizure is about 70% within the first year, and most recurrent attacks occur within a month or two of the first.
5. Further seizures are loss likely if a trigger factor is definable and avoidable.
6. In some conditions epilepsy is the only feature of underlying disease, whilst in others epilepsy is just one of the manifestations.
7. The annual incidence of new cases of epilepsy after infancy is 2070/100 000.
8. The lifetime risk of having a single seizure is about 5% whilst the prevalence of epilepsy in European countries is about 0.5%.
9. The prevalence in developing countries is up to five times higher than in developed countries.

Classification

The classification of epilepsy is best achieved by considering the clinical events (the seizure), the abnormal electrophysiology, the anatomical site of seizure genesis and the pathological cause of the problem.

Classification of epilepsy

Seizure type :

- Partial (simple or complex)
- Tonic clonic
- Partial with secondary generalisation
- Tonic
- Atonic
- Absence
- Myoclonic

Physiology (EEG)

- Focal spikes/sharp waves Anatomical site
- Generalised spike and wave
- Cortex
  Temporal
  Frontal
  Parietal
  Occipital
- Generalised (diencephalon)
- Multifocal

Pathological cause :

- Genetic
- Developmental
- Tumours
- Trauma
- Vascular
- Infections
- Inflammation
- Metabolic
- Drugs, alcohol and toxins
- Degenerative

A. Primary generalised epilepsy

- The primary of idiopathic epilepsis makes up some 10% of all epilepsis, including some 40% of those with tonic clonic seizures
- Onset is almost always in childhood or adolescence.
- No structural abnormally is present and there is often a substantial genetic predisposition.
- Some like childhood absence epilepsy, are relatively uncommon, whilst others, like juvenile with epilepsy

The more common varieties of primary generalised epilepsy along with their clinical features and management.

Primary generalised epilepsies

	Incidence	Age of onset	Type of	EEG features Seizure	Provoking	Treatment factors	Prognosis
Childhood absences epilepsy	6-8/100000	4-8 yrs	Frequent brief absence	3/s spike and wave	Hyperventilation, fatigue	Ethosuximide Sodium valproate	40% develop GTCS, 80% remit in adulthood
Juvenile absence epilepsy	1-2/100000	10-15 yrs	Less frequent absences than childhood absence	Poly-spike and wave	Hyperventilation, sleep deprivation	Sodium valproate	80% develop GTCS, 80% seizure-free in adulthood
Juvenile myoclonic epilepsy	25-50/100 000	15-20 yrs	GTCS, absences morning myoclonus	Poly-spike and wave, photosensitivity	Sleep deprivation, alcohol withdrawal	Sodium valproate	90% remit with AEDs but relapse if AED withdrawn
GTCS on awakening	Common	10-25 yrs	GTCS, sometimes myoclonus	'Spike and Wave on waking and sleep onset	Sleep deprivation	Sodium valproate	65% Controlled with AEDs but relapse off treatment

(GTCS = generalised tonic clonic seizure; AED = anti-epileptic drug)

B. Partial epilepsy

Partial seizures may arise from any disease of the cerebral cortex, congenital or acquired and frequently generalise with the exceptions of a few idiopathic partial epilepsies of benign outcome in childhood, the presence of partial seizures signifies the presence of focal cerebral pathology.

Causes of partial seizures

Idiopathic

- Benign Rolandic epilepsy of childhood
- Benign occipital epilepsy of childhood

Focal structural lesions

Genetic

- Tuberous sclerosis
- Neurofibromatosis
- von Hippel-Lindau disease
- Cerebral migration abnormalities

Infantile hemiplegia

Dysembryonic

- Cortical dysgenesis
- Sturge-Weber syndrome

Mesial temporal sclerosis (associated with febrile convulsions)

Cerebrovascular disease

- Intracerebral haemorrhage
- Arteriovenous malformation
- Cerebral infarction
- Cavernous haemangioma

Tumours (primary and secondary)

Trauma (including neurosurgery)

Infective

- Cerebral abscess (pyogenic)
- Subdural empyema
- Toxoplasmosis
- Encephalitis
- Cysticercosis
- Human immunodeficiency virus (HIV)
- Tuberculoma

Inflammatory

- Sarcoidosis
- Vasculitis

Secondary generalised epilepsy

1. Generalised epilepsy may arise from spread of partial seizures due to structural disease, or may be secondary to drugs or metabolic disorders.
2. Epilepsy presenting in adult life is almost always secondary generalised, even if there is no clear history of a partial seizure before the onset of a major attack (an 'aura').

Causes of secondary generalised epilepsy

Secondary generalisation from partial seizures

- All causes of partial seizures

Genetic

- Inborn errors of metabolism
- Storage diseases
- Phakomatoses (e.g. tuberous sclerosis)

Cerebral birth injury

Hydrocephalus

Cerebral anoxia

Drugs

- Antibiotics: penicillin, isoniazid, metronidazole
- Antimalarials: chloroquine, mefloquine
- Ciclosporin
- Cardiac anti-arrhythmics: lidocaine, disopyramide
- Psychotropic agents: phenothiazines, tricyclic antidepressants, lithium
- Amphetamines (withdrawal)

Alcohol (especially withdrawal)

Toxins

- Heavy metals (lead, tin)
- Organophosphates (sarin)

Metabolic disease

- Hypocalcaemia
- Hypoglycaemia
- Hyponatraemia
- Renal failure
- Hypomagnesaemia
- Liver failure Infective
- Meningitis
- Post-infectious encephalopathy

Inflammatory

- Multiple sclerosis (uncommon)
- SLE

Diffuse degenerative diseases

- Alzheimer's disease (uncommonly)
- Creutzfeldt-Jakob disease (rarely)

Pathogenesis

1. The occurrence of epilepsy in those over the age of 60 is frequently inhibitory transmitter gamma-aminobutyric acid (GABA) is particularly important in this role, and drugs that block GABA receptors provoke seizures.
2. Conversely, excessive stimulation by excitatory neurotransmitters, such as acetylcholine, glutamate and aspartate, provoke seizure activity.
3. It is likely that both reduction of inhibition and

excessive excitation play a part in the genesis of most seizures.

4. The cerebral cortex in epilepsy exhibits hypersynchronous, repetitive discharges involving large groups of neurons, intracellular recordings of which demonstrate burst of high-frequency action potentials associated with a reduction in the transmembrane potential (paroxysmal depolarization shift).

Clinical features

1. When taking a history from a patient with suspected seizure, it is important to clarify which type of attack is occurring, bearing in mind that there may be more than one occurring in the same patient at different times.
2. Sometimes specific trigger factors can be identified namely
   - Sleep deprivation
   - Alcohol (particularly withdrawal)
   - Recreational drug misuse
   - Physical and mental exhaustion
   - Flickering lights, including TV and computer screens (primary generalised epilepsies only)
   - Intercurrent infections and metabolic disturbances
   - Uncommonly: loud noises, music, reading, hot baths

Tonic clonic seizures

1. A tonic clonic seizure may be preceded by a partial seizure (the'aura') which can take various forms
2. The patient then goes rigid and becomes unconscious, falling down heavily if standing and often sustaining injury.
3. During this phase respiration is arrested and central cyanosis may occur.
4. After a few moments, the rigidity is periodically relaxed, producing clonic jerks.
5. Some patients do not have a clonic phase and the rigidity is replaced by a flaccid state of deep coma which can persist for some minutes.
6. The patient then gradually regains consciousness, but is in a confused and disorientated state for half an hour or more after regaining consciousness.
7. Full memory function may not be recovered for some hours.
8. During the attack, urinary incontinence and tongue-biting may occur.
9. A severely bitten, bleeding tongue after an attack of loss of consciousness is pathognomonic of a generalised seizure.
10. After a generalised seizure the patient usually feels terrible, may have a headache and will often want to sleep.

Complex partial seizures

1. Complex partial seizures involve episodes of altered consciousness without the patient collapsing to the ground, especially if they arise from the temporal or, less frequently, the frontal lobe.
2. Patients stop what they are doing and stare blankly, often blinking repetitively, making smacking movements of their lips, or displaying other automatisms, such as picking at their clothes.
3. After a few minutes, consciousness returns but the patient may be muddled and feel drowsy for a period of up to an hour.
4. Immediately before such an attack the patient may report alteration of mood, memory or perception (such as undue familiarity-deja vu- or unreality-jamais vu), or complex hallucinations involving sound, smell, taste, vision, emotion (fear, sexual arousal) or visceral sensations (nausea, epigastric discomfort)

Simple partial' seizure

1. Changes in memory or perception occur without subsequent alteration in awareness, the seizure is said to be a 'simple partial' seizure.
2. Complex partial seizures arising from the anterior parts of the frontal lobe may produce bizarre behaviour patterns including limb posturing, sleep walking, or even frenetic ill-

directed motor activity with incoherent screaming.

3. These can sometimes be very difficult to distinguish from psychogenic attacks (which are more common).
4. However, abruptness of onset and relative brevity may help point to frontal seizures, as may a tendency to occur out of sleep.

Absence seizures

1. Absence seizures (petit mal) always start in childhood.
2. The attacks can be mistaken for complex partial seizures but are shorter in duration
3. Occur much more frequently (20-30 times a day) and are not associated with post-ictal confusion.
4. Absence attacks are caused by a generalised discharge that does not spread out of the hemispheres and so does not cause loss of posture.

Atonic seizures

1. These are seizures involving brief loss of muscle tone, usually resulting in heavy falls with or without loss of consciousness.
2. They only occur in the context of epilepsy syndromes which involve other forms of seizure. They are not a cause of collapse in patients without epilepsy.

Partial motor seizures

1. Epileptic activity arising in the pre-central gyrus causes partial motor seizures affecting the contralateral face, arm, trunk or leg.
2. Seizures are characterised by rhythmical jerking or sustained spasm of the affected parts.
3. They may remain localised to one part, or may spread to involve the whole side.

Jacksonian seizure

1. Some attacks begin in one part of the body (e.g. mouth, thumb, great toe) and spread (march) gradually to other parts of the body; this is known as a Jacksonian seizure.
2. Attacks vary in duration from a few seconds to several hours (epilepsia partialis continua).
3. More prolonged episodes may be followed by paresis of the involved limb lasting for several hours after the seizure ceases (Todd's palsy).

Partial sensory seizures

1. Seizures arising in the sensory cortex cause unpleasant tingling or 'electric' sensations in the contralateral face or limbs.
2. A spreading pattern similar to a Jacksonian seizure may occur, the abnormal sensation spreading over the body much faster (in seconds) than the march of a migrainous focal sensory attack, which spreads over 20-30 minutes.

Versive seizures

1. A frontal epileptic focus may involve the frontal eye field, causing forced deviation of the eyes and sometimes turning of the head to the opposite side.
2. This type of attack often generalises to become a tonic clonic seizure.

Partial visual seizures

1. Occipital epileptic foci cause simple visual hallucinations such as balls of light or patterns of colour.
2. Formed visual hallucinations of faces or scenes arise more interiorly in the temporal lobes.

Investigations

Are the attacks truly epileptic?

- Ambulatory EEG
- Videotelemetry

From where is the epilepsy arising?

- Standard EEG
- Sleep EEG
- EEG with special electrodes (foramen ovale, subdural)

What is the cause of the epilepsy?

Structural lesion?

- CT
- MRI

Metabolic disorder?

- Urea and electrolytes

- Liver function tests
- Blood glucose
- Serum calcium, magnesium Inflammatory or infective disorder?
- Full blood count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)
- Chest X-ray
- Serology for syphilis, HIV, collagen disease
- CSF examination

Indications for brain imaging in epilepsy

- Epilepsy starting after the age of 20 years
- Seizures having focal features clinically
- EEG showing a focal seizure source
- Control of seizures difficult or deteriorating

Epilespsy in old age

- Incidence and prevalence: late-onset epilepsy is very common and the annual incidence in those over 60 years is rising.
- Fits and faints: the features that usually differentiate these may be less definitive than in younger patients.
- Complex partial status epilepticus: can present as confusion in the elderly.
- Cerebrovascular disease is the underlying cause of seizures in 30-50% of patients over the age of 50 years. A seizure may occur with an overt stroke or with occult vascular disease.
- Anti-epileptic drug regimens: keep as simple as possible and take care to avoid interactions with other drugs being prescribed.
- Carbamazepine-induced hyponatraemia: increases significantly with age; particularly important in patients on diuretics or those with heart failure.

MCQs

1. For the diagnoses of depression, symptoms must be present for at least

a. 2 weeks	b. 1 month
c. 2 months	d. 6 months

2. Anxiety Neurosis is diagnosed by the following features
   - a. Excessive, uncontrollable worry
   - b. Excessive, uncontrollable worry + 3 additional anxiety symptoms most days, for atleast 6 months.
   - c. Excessive, uncontrollable worry + 1 additional anxiety symptoms, for at least 2 weeks.
   - d. Persistent feelings of sadness, hopelessness+ 1 additional anxiety symptoms, for at least 2 weeks
3. Recurrentsuicidal ideation seen in
   - a. Anxiety Disorders
   - b. Bipolar Disorder
   - c. Depression
   - d. Post-Traumatic Stress Disorder

Answer

1. a	2. b	3. c

CHAPTER
54

Phiranga and Upadamsha

Prof. Parameswarappa S. Byadgi and Dr. Anurag Pandey

फिरङ्ग (Phiraṅga)

Introduction

A kind of Sexually transmitted disorder. *Upadamśa* (syphilis) not treated properly may become generalized through lymphatics affecting skin, mucosa and nearly all the tissues of the body and this indisposition is identified as *Phiraṅga*. The term has derived from the English word foreign.

Definition

फिरङ्गसंज्ञके देशे बाहुल्येनैव यद्भवेत् ।
तस्मात् फिरङ्ग इत्युक्तो व्याधिर्व्याधिविशारदैः ॥

BPM. 59/1

It is predominant in the country named *Phiraṅga* (Portugal) hence it is termed as *Phiraṅga* by the physicians.

फिरङ्ग निदान (Phiraṅga Nidāna)

गन्धरोगः फिरङ्गोऽयं जायते देहिनां ध्रुवम् ।
फिरङ्गिणोऽङ्गसंसर्गात्फिरङ्गिण्याः प्रसङ्गतः ॥

BPM 59/2

Phiraṅga is also known as *Gandha roga*, manifest by the touch of persons of *Phiraṅga Deśa* and having sexual contact with Portugese women.

फिरङ्ग संप्राप्ति (Phiraṅga Samprāpti)

व्याधिरागन्तुजो ह्येष दोषाणामत्र सङ्क्रमः।
भवेत्तं लक्षयेत्तेषां लक्षणैर्भिपजां वरः॥

BPM 59/3

Vitiations of *Doṣās* develop later because it is a exogenous disease & has to be diagnosed by the physician from symptoms which appear.

फिरङ्ग लक्षण (Phiraṅga Lakṣaṇa)

फिरङ्गस्त्रिविधो ज्ञेयो बाह्य आभ्यन्तरस्तथा ।
बहिरन्तर्भवश्चापि तेषां लिङ्गानि च ब्रुवे ॥
तत्र बाह्यः फिरङ्गः स्याद्विस्फोटसदृशोऽल्परुक् ।
स्फुटितो व्रणवद्वेद्यः सुखसाध्योऽपि स स्मृतः ॥
सन्धिष्वाभ्यन्तरः स स्यादामवात इव व्यथाम् ।
शोथञ्च जनयेदेष कष्टसाध्यो बुधैः स्मृतः ॥

BPM 59/4-6

Phiraṅga is classified into three varieties i:e (i) *Bāhya* (ii) *Abhyantara* & (iii) *Bahirantarbhava.*

बाह्य फिरङ्ग (Bāhya Phiraṅga)

External *Phiraṅga* has vesicle associated with mild pain. When it bursts gives rise to an ulcer and can be cured easily.

अभ्यन्तर फिरङ्ग (Abhyantara Phiraṅga)

It manifest in the joints & produces pain similar to *Āmavāta* & it is very difficult to cure.

फिरङ्ग उपद्रव (Phiraṅga Upadrava)

कार्श्यं बलक्षयो नासाभङ्गो वह्नेश्च मन्दता ।
अस्थिशोषोऽस्थिवक्रत्वं फिरङ्गोप्रद्रवा अमी ॥

BPM 59/7

Complications of *Phiraṅga* are emaciation, loss of strength, mutilation of the nose, depletion of digestive power, dryness & deformities of the bones.

साध्यासाध्यता (Sādhyasādhyatā)

बहिर्भवो भवेत्साध्यो नवीनो निरुपद्रवः ।
आभ्यन्तरस्तु कष्टेन साध्यः स्यादयमामयः ॥
बहिरन्तर्भवो जीर्णः क्षीणस्योपद्रवैर्युतः ।
व्याप्तो व्याधिरसाध्योऽयमित्याहुर्मुनयः पुरा ॥

BPM 59/8-9

Phiraṅga which is external, newly manifested without complications is easily curable. *Phiraṅga* which is internal curable with difficulty. *Phiraṅga* which is both internal & external, chronic, emaciated persons & spreaded to other body parts is incurable.

उपदंश (Upadamśa)

Introduction

Veneral Diseases. Factors which lead to veneral diseases (*Upadamśa*) includes the following: injury to the penis by the hand, nails and teeth; keeping it dirty without washing, indulging in excessive sexual intercourse, contact with contaminated or diseased vagina and such other bad habits and it is of five types.

उपदंश निदान (Upadamśa Nidāna)

हस्ताभिघातान्नखदन्तघातादधावनादत्युपसेवनाद्वा ।
योनिप्रदोषाच्च भवन्ति शिश्ने पञ्चोपदंशा विविधापचारैः ॥
BPM. 51/1

Trauma to the penis either by the hand nails & teeth; not cleaning the penis properly; indulging in excessive sexual intercourse; contact with diseased vagina and many other abnormal activities gives rise to five varieties of *Upadamśa*.

तत्रातिमैथुनादतिब्रह्मचर्य्याद्वा तथाऽतिब्रह्मचारिणीं चिरोत्सृष्टां रजस्वलां दीर्घरोमां कर्कशरोमां सङ्कीर्णरोमां निगूढरोमामल्पद्वारां महाद्वारामप्रियामकामाम् चौक्ष्यसलिलप्रक्षालित योनिमप्रक्षालितयोनिं यो निरोगोपसृष्टां स्वभावतो वा दुष्टयोनिं वियोनिं वा नारीमत्यर्थमुपसेवमानस्य तथा करजदशनविषशूकनिपातनाद्बन्धनाद्धस्ताभिघाताच्चतुष्पदीगमनादचौक्ष्यसलिलप्रक्षालनादवपीडनाच्छुक्रमूत्रवेगविधारणान्मैथुनान्ते वाऽप्रक्षालनादिभिर्मेढ्रमागम्य प्रकुपिता दोषाः क्षतेऽक्षते वा श्वयथुमुपजनयन्ति, तमुपदंशमित्याचक्षते ॥ *S.S.Ni. 12/7*

Etiological factors are as follows : Person who is indulging in too much sexual intercourse or too much celibacy, copulating with the woman who is most celibate, who has avoided sexual intercourse for longer period, who is in her menstrual period, whose pubic hairs are long, rough, matted together or hidden; whose vaginal orifice is small or big; who is not liked; not sensual; who has washed the vagina with contaminated water or not washed at all; who is suffering from diseases of vagina; he who uses other places (mouth, rectum, armpits etc.), injury by nails, teeth, application of paste of poisonous insects & by wrapping it, squeezing by hands, sexual intercourse with quadrupeds (cow, goat, sheep, donkey, horse, dog etc.), washing the penis with dirty water, vigorous kneading, controlling ejalulation, not washing the penis soon after sexual intercourse, & such other abnormal activities aggravates the *Doṣās* in the penis and causes swelling either with a wound or without is known as *Upadamśa*.

उपदंश भेद (Upadamśa Bheda)

Upadamśa is classified into 5 varieties namely :

(i) *Vātaja Upadamśa*
(ii) *Pittaja Upadamśa*
(iii) *Raktaja Upadamśa*
(iv) *Kaphaja Upadamśa*
(v) *Tridoṣaja Upadamśa.*

उपदंश लक्षण (Upadamśa Lakṣaṇa)

सतोदभेदस्फुरणैः सकृष्णैः स्फोटर्व्यवस्येत्पवनोपदंशम् ।
पीतैर्बहुक्लेदयुतः सदाहैः पित्तेन रक्तैः पिशितावभासैः ॥
स्फोटैः सकृष्णै रुधिरं स्रवन्तं रक्तात्मकं पित्तसमानलिङ्गम् ।
सकण्डुरैः शोथयुतैर्महद्भिः शुक्लैर्घनैः स्रावयुतैः कफेन ।
नानाविधस्रावरुजोपपन्नमसाध्यमाहुस्त्रिमलोपदंशम् ॥
BPM. 51/2-3

वातज उपदंश (Vātaja Upadamśa)

तत्र वातिके पारुष्यं त्वक्परिपुटनं स्तब्धमेढ्रता परुषशोफता विविधाश्च वातवेदनाः । *S.S.Ni. 12/12*

It is characterised by papules in the penis, which is black associated with pricking or pulsating pain, penis has no movements, coarse to touch, & has different kinds of pain of *vāta* origin.

पित्तज उपदंश (Pittaja Upadamśa)

पैत्तिके ज्वरः श्वयथुः पक्वोदुम्बरसङ्काशस्तीव्रदाहः क्षिप्रपाकः पित्तवेदनाश्च । *S.S.Ni. 12/12*

It is characterised by papules in the penis, which is yellow associated with more exudation & burning sensation, fever, swelling resembles a ripe fruit of *Udumbara*, suppurates quickly, & has different kinds of pain of *Pitta* origin.

कफज उपदंश (Kaphaja Upadamśa)

श्लेष्मिके श्वयथुः कण्डूमान् कठिनः स्निग्धः श्लेष्मवेदनाश्च ।
S.S.Ni. 12/9

It is characterised by papules in the penis which is big associated with itching, exudation of thick, white fluid, hard, unctuous & has different kinds of pain of *Kaphaja* origin.

रक्तज उपदंश (Raktaja Upadamśa)

रक्तजे कृष्णस्फोटप्रादुर्भावोऽत्यर्थमसृक्प्रवृत्तिः पित्तलिङ्गान्यत्यर्थं ज्वरदाहौ शोषश्च याप्यश्चैव कदाचित् । *S.S.Ni. 12/12*

It is characterised by the papules in the skin which resemble muscles, blackish, exuding blood and all other symptoms of *Pittaja Upadamśa*. It is only manageable disease.

त्रिदोषज उपदंश (Tridoṣaja Upadamśa)

सर्वजे सर्वलिङ्गदर्शनमवदरणं च शेफसः कृमिप्रादुर्भावो मरणं चेति । *S.S.Ni. 12/9*

It is characterised by appearance of symptoms of all three *Doṣās*. Papules appear in the penis, which discharges different kinds of exudates, manifest various kinds of pain, penis become cracked, associated with *Krimi* & *kills* the person & it is incurable.

साध्यासाध्यता (Sādhyāsādhyatā)

प्रशीर्णमासं कृमिभिः प्रजग्धं मुष्कावशेषं परिवर्जनीयम् ।
सञ्जातमात्रे न करोति मूढः क्रियां नरो यो विषये प्रसक्तः ।
कालेन शोथकृमिदाहपाकैः प्रशीर्णशिश्नो म्रियते स तेन ॥
BPM 51/4-5

The foolish person who does not seek proper treatment soon after its onset cause the development of *Krimi,* which starts eating the *Māmsa* of the penis leaving only the scrotum, such condition should be rejected for treatment.

Due to negligence the patient become victims of swelling, infection, burning sensation, suppuration, destruction of penis & death.

उपशय/पथ्य (Upaśaya/Pathya)

The following measures and edibles are considered to be helpful for the patients of *Upadamśa*: *Vamana*, *Virecana*, operating (*Vedhana*) the penis middle vein, blood letting through leeches, the *Secana* therapy, ointmenting, barley, *Śāli* type of paddy, meat of wild animals and birds, *Yūṣa* of *Mudga*, *Ghṛtam*, *Kāravellaka*, fruits of *Śobhāñjana*, *Paṭola*, *Śāliañca* leaves, young radish, edibles having bitter and pungent tastes, honey, water from the well and sesame oil. (B.R. 52/89-90)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures and edibles are to be avoided by a patient of *Upadamśa*: sleeping during day hours, obstructing flow of urine, heavy to digest meals, intercourse, raw sugar (*Guḍa*), performing labourious tasks, taking citrus edibles and buttermilk. (B.R. 52/91)

MCQs

1. Types of *Upadamsa*
   a. 3 b. 5
   c. 7 d. 9
2. *Krimi* seen in
   a. *Kaphaja Upadamsa*
   b. *Paithika Upadamsa*
   c. *Raktaja Upadamsa*
   d. *Tridoshaja Upadamsa*
3. Prognosis of *Raktaja Upadamsa*
   a. *Asadya* b. *Krichra sadya*
   c. *Yapya* d. *Dussadya*
4. *Gandha roga is the synonym of*
   a. *Upadamsa* b. *Phiranga*
   c. *Nadivrana* d. *Masurika*
5. *Prognosis of Abhyanthara Phiranga*
   a. *Yapya* b. *Kashta sadya*
   c. *Dussadya* d. *Anupakrama*
6. *Nasabhanga is the Upadrava of*
   a. *Phiranga* b. *Upadamsa*
   c. *Asthi vidradi* d. *Slipada*

Answer

1.	b	2.	d	3.	c
4.	b	5.	b	6.	a

CHAPTER

55

Syphilis and Gonorrhoea

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

Syphilis

Syphilis, a chronic systemic infection caused by Treponemapallidum subspecies pallidum, is usually sexually transmitted and is characterized by episodes of active disease interrupted by periods of latency. After an incubation period averaging 2-6 weeks, a primary lesion appears, often associated with regional lymphadenopathy. The secondary stage, associated with generalized mucocutaneous lesions and generalized lymphadenopathy, is followed by a latent period of subclinical infection lasting years or decades. Central nervous system (CNS) involvement may occur early in infection and may be symptomatic or asymptomatic. In about one-third of untreated cases, the tertiary stage appears, characterized by progressive destructive mucocutaneous, musculoskeletal, or parenchymal lesions; aortitis; or late CNS manifestations.

Etiology

The *Treponema* species include *T. pallidum* subspecies *pallidum*, which causes venereal syphilis.

Transmission and Epidemiology

Nearly all cases of syphilis are acquired by sexual contact with infectious lesions [i.e., the chancre, mucous patch, skin rash, or condylomatalata. Less common modes of transmission include nonsexual personal contact, infection in utero, blood transfusion, and organ transplantation.

Clinical Manifestations

Primary Syphilis : The typical primary chancre usually begins as a single painless papule that rapidly becomes eroded and usually becomes indurated, with a characteristic cartilaginous consistency on palpation of the edge and base of the ulcer. Multiple primary lesions are seen in a minority of patients. In heterosexual men the chancre is usually located on the penis, whereas in homosexual men it may be found in the anal canal or rectum, in the mouth, or on the external genitalia. In women, common primary sites are the cervix and labia. Consequently, primary syphilis goes unrecognized in women and homosexual men more often than in heterosexual men. Atypical primary lesions are common. The clinical appearance depends on the number of treponemes inoculated and on the immunologic status of the patient. A large inoculum produces a dark-field-positive ulcerative lesion in nonimmune volunteers but may produce a small dark-field-negative papule, an asymptomatic but seropositive latent infection, or no response at all in some individuals with a history of syphilis. A small inoculum may produce only a papular lesion, even in nonimmune individuals. Therefore, syphilis should be considered even in the evaluation of trivial or atypical dark-field-negative genital lesions. The genital lesions that most commonly must be differentiated from those of primary syphilis include those caused by herpes simplex virus infection, chancroid, traumatic injury, and donovanosis. Regional (usually inguinal) lymphadenopathy accompanies the primary syphilitic lesion, appearing within 1 week of lesion onset. The nodes are firm, nonsuppurative, and painless. Inguinal lymphadenopathy is bilateral and may occur with anal as well as with external genital chancres. The chancre generally heals within 4-6 weeks (range, 2-12 weeks), but lymphadenopathy may persist for months.

Secondary Syphilis

The protean manifestations of the secondary stage usually include mucocutaneous lesions and generalized nontender lymphadenopathy. The healing primary chancre may still be present in 15% of cases, and the stages may overlap more frequently in persons with concurrent HIV infection. The skin rash consists of macular, papular, papulosquamous, and occasionally pustularsyphilides; often more than one form is present simultaneously. The eruption may be very subtle, and 25% of patients with a discernible rash may be unaware that they have dermatologic manifestations. Initial lesions are pale red or pink, nonpruritic, discrete macules distributed on the trunk and proximal extremities; these macules progress to papular lesions that are distributed widely and that frequently involve the palms and soles. Rarely, severe necrotic lesions (luesmaligna) may appear; they are more commonly reported in HIV-infected individuals. Involvement of the hair follicles may result in patchy alopecia of the scalp hair, eyebrows, or beard in up to 5% of cases.

In warm, moist, intertriginous areas (commonly the perianal region, vulva, and scrotum), papules can enlarge to produce broad, moist, pink or gray-white, highly infectious lesions [condylomatalata (see Fig. e7-20)] in 10% of patients with secondary syphilis. Superficial mucosal erosions (mucous patches) occur in 10-15% of patients and commonly involve the oral or genital mucosa. The typical mucous patch is a painless silver-gray erosion surrounded by a red periphery.

Constitutional symptoms that may accompany or precede secondary syphilis include sore throat (15-30%), fever (5-8%), weight loss (2-20%), malaise (25%), anorexia (2-10%), headache (10%), and meningismus (5%). Acute meningitis occurs in only 1-2% of cases, but CSF cell and protein concentrations are increased in up to 40% of cases, and T. pallidum has been recovered from CSF during primary and secondary syphilis in 30% of cases; the latter finding is often but not always associated with other CSF abnormalities.

Less common complications of secondary syphilis include hepatitis, nephropathy, gastrointestinal involvement (hypertrophic gastritis, patchy proctitis, or a rectosigmoid mass), arthritis, and periostitis. Ocular findings that suggest secondary syphilis include pupillary abnormalities and optic neuritis as well as the classic iritis or uveitis. The diagnosis of secondary syphilis is often considered in affected patients only after they fail to respond to steroid therapy. Anterior uveitis has been reported in 5-10% of patients with secondary syphilis, and T. pallidum has been demonstrated in aqueous humor from such patients. Hepatic involvement is common in syphilis; although usually asymptomatic, up to 25% of patients may have abnormal liver function tests. Frank syphilitic hepatitis may be seen. Renal involvement usually results from immune complex deposition and produces proteinuria associated with an acute nephrotic syndrome. Like those of primary syphilis, the manifestations of the secondary stage resolve spontaneously, usually within 1-6 months.

Latent Syphilis

Positive serologic tests for syphilis, together with a normal CSF examination and the absence of clinical manifestations of syphilis, indicate a diagnosis of latent syphilis in an untreated person. The diagnosis is often suspected on the basis of a history of primary or secondary lesions, a history of exposure to syphilis, or the delivery of an infant with congenital syphilis. A previous negative serologic test or a history of lesions or exposure may help establish the duration of latent infection, which is an important factor in the selection of appropriate therapy. Early latent syphilis is limited to the first year after infection, whereas late latent syphilis is defined as that of 1 year's (or unknown) duration. T. pallidum may still seed the bloodstream intermittently during the latent stage, and pregnant women with latent syphilis may infect the fetus in utero. Moreover, syphilis has been transmitted through blood transfusion or organ donation from patients with latent syphilis. It was previously thought that untreated late latent syphilis had three possible outcomes: (1) persistent lifelong infection; (2) development of late syphilis; or (3) spontaneous cure, with reversion of serologic tests to negative. It is now apparent, however, that the more sensitive treponemal antibody tests rarely, if ever, become negative without treatment. Although progression to clinically evident late syphilis is very rare today, the occurrence of spontaneous cure is in doubt.

Involvement of the CNS

Traditionally, neurosyphilis has been

considered a late manifestation of syphilis, but this view is inaccurate. CNS syphilis represents a continuum encompassing early invasion (usually within the first weeks or months of infection), months to years of asymptomatic involvement, and, in some cases, development of early or late neurologic manifestations.

Other Manifestations of Late Syphilis

The slowly progressive inflammatory disease leading to tertiary disease begins early during infection, although these manifestations may not become clinically apparent for years or decades. Early syphilitic aortitis becomes evident soon after secondary lesions subside, and treponemes that trigger the development of gummas may have seeded the tissue years earlier.

Cardiovascular Syphilis

Cardiovascular manifestations, usually appearing 10-40 years after infection, are attributable to endarteritis obliterans of the vasa vasorum, which provide the blood supply to large vessels; T. pallidum DNA has been detected by PCR in aortic tissue. Cardiovascular involvement results in uncomplicated aortitis, aortic regurgitation, saccular aneurysm (usually of the ascending aorta), or coronary ostial stenosis. In the preantibiotic era, symptomatic cardiovascular complications developed in 10% of persons with late untreated syphilis, although syphilitic aortitis was demonstrated at autopsy in about one-half of African-American men with untreated syphilis. Today, this form of late syphilis is rarely seen in the developed world. Linear calcification of the ascending aorta on chest x-ray films suggests asymptomatic syphilitic aortitis, as arteriosclerosis seldom produces this sign. Syphilitic aneurysms-usually saccular, occasionally fusiform do not lead to dissection. Only 1 in 10 aortic aneurysms of syphilitic origin involves the abdominal aorta.

Late Benign Syphilis (Gumma)

Gummas are usually solitary lesions ranging from microscopic to several centimeters in diameter. Histologic examination shows a granulomatous inflammation, with a central area of necrosis due to endarteritis obliterans. Although rarely demonstrated microscopically, T. pallidum has been detected by PCR or recovered from these lesions, and penicillin treatment results in rapid resolution, confirming the treponemal stimulus for the inflammation. Common sites include the skin and skeletal system; however, any organ (including the brain) may be involved. Gummas of the skin produce indolent, painless, indurated nodular or ulcerative lesions that may resemble other chronic granulomatous conditions, including tuberculosis, sarcoidosis, leprosy, and deep fungal infections. Skeletal gummas most frequently involve the long bones, although any bone may be affected. Upper respiratory gummas can lead to perforation of the nasal septum or palate.

Congenital Syphilis

Transmission of T. pallidum across the placenta from a syphilitic woman to her fetus may occur at any stage of pregnancy, but fetal damage generally does not occur until after the fourth month of gestation, when fetal immunologic competence begins to develop. This timing suggests that the pathogenesis of congenital syphilis, like that of adult syphilis, depends on the host immune response rather than on a direct toxic effect of T. pallidum. The risk of fetal infection during untreated early maternal syphilis is 75-95%, decreasing to 35% for maternal syphilis of >2 years' duration. Adequate treatment of the woman before the 16th week of pregnancy should prevent fetal damage, and treatment before the third trimester should adequately treat the infected fetus. Untreated maternal infection may result in a rate of fetal loss of up to 40% (with stillbirth more common than abortion because of the late onset of fetal pathology), prematurity, neonatal death, or nonfatal congenital syphilis. Among infants born alive, only fulminant congenital syphilis is clinically apparent at birth, and these babies have a very poor prognosis. The most common clinical problem is the healthy-appearing baby born to a mother with a positive serologic test. Routine serologic testing in early pregnancy is considered cost-effective in virtually all populations, even in areas with a low prenatal prevalence of syphilis. Low-tech point-of-care tests are being developed to facilitate antenatal testing in resource-poor settings. Where the prevalence of syphilis is high or when the patient is at high risk of re-infection, serologic testing should be repeated in the third trimester and at delivery. Neonatal congenital syphilis must be differentiated from other generalized congenital infections, including rubella, cytomegalovirus or herpes simplex virus infection, and toxoplasmosis, as well as from erythroblastosisfetalis.

The manifestations of congenital syphilis include (1) early manifestations, which appear within the first 2 years of life (often at 2-10 weeks of age), are infectious, and resemble the manifestations of secondary syphilis in the adult; (2) late manifestations, which appear after 2 years and are noninfectious; and (3) residual stigmata. The earliest manifestations of congenital syphilis (appearing 2-6 weeks after birth) include rhinitis, or "snuffles" (23%); mucocutaneous lesions (35-41%); bone changes (61%), including osteochondritis, osteitis, and periostitis detectable by x-ray examination of long bones; hepatosplenomegaly (50%); lymphadenopathy (32%); anemia (34%); jaundice (30%); thrombocytopenia; and leukocytosis. CNS invasion by T. pallidum is detectable in 22% of infected neonates. Neonatal death is usually due to pulmonary hemorrhage, secondary bacterial infection, or severe hepatitis.

Late congenital syphilis (untreated after 2 years of age) is subclinical in 60% of cases; the clinical spectrum in the remainder of cases may include interstitial keratitis (which occurs at 5-25 years of age), eighth-nerve deafness, and recurrent arthropathy. Bilateral knee effusions are known as Clutton's joints. Asymptomatic neurosyphilis is present in about one-third of untreated patients, and clinical neurosyphilis occurs in one-quarter of untreated individuals >6 years old. Gummatouperiostitis occurs at 5-20 years of age and, as in nonvenereal endemic syphilis, tends to cause destructive lesions of the palate and nasal septum.

Classic stigmata include Hutchinson's teeth (centrally notched, widely spaced, peg-shaped upper central incisors), "mulberry" molars (sixth-year molars with multiple, poorly developed cusps), saddle nose, and saber shins.

Ancillary Investigations

Laboratory Examinations

Demonstration of the Organism : T. pallidum cannot be detected by culture. Historically, dark-field microscopy and immunofluorescence antibody staining have been used to identify this spirochete in samples from moist lesions such as chancres or condylomatalata, but these tests are rarely available today outside of research laboratories. More sensitive PCR tests have been developed but are not commercially available, although some laboratories perform in-house PCR testing.

T. pallidum can be found in tissue with appropriate silver stains, but these results should be interpreted with caution because artifacts resembling T. pallidum are often seen. Tissue treponemes can be demonstrated more reliably in research laboratories by PCR or by immunofluorescence or immunohistochemical methods using specific monoclonal or polyclonal antibodies to T. pallidum.

Serologic Tests for Syphilis

There are two types of serologic test for syphilis: nontreponemal and treponemal. Both are reactive in persons with any treponemal infection, including yaws, pinta, and endemic syphilis.

The most widely used nontreponemal antibody tests for syphilis are the rapid plasma reagin (RPR) and Venereal Disease Research Laboratory (VDRL) tests, which measure IgG and IgM directed against a cardiolipin-lecithin-cholesterol antigen complex. The RPR test is easier to perform and uses unheated serum; it is the test of choice for rapid serologic diagnosis in a clinical setting and can be automated. The VDRL test remains the standard for examining CSF. The RPR and VDRL tests are recommended for screening or for quantitation of serum antibody. The titer reflects disease activity, rising during the evolution of early syphilis and often exceeding 1:32 in secondary syphilis. After therapy for early syphilis, a persistent fall by fourfold or more (e.g., a decline from 1:32 to 1:8) is considered an adequate response. VDRL titers do not correspond directly to RPR titers, and sequential quantitative testing (as for response to therapy) must employ a single test.

Treponemal tests measure antibodies to native or recombinant T. pallidum antigens and include the fluorescent treponemal antibody-absorbed (FTA-ABS) test and the T. pallidum particle agglutination (TPPA) test, both of which are more sensitive for primary syphilis than the previously used hemagglutination tests. The T. pallidumhemagglutination (TPHA) test is widely used in Europe but is not available in the United States. When used to confirm positive nontreponemal test results, treponemal tests have a very high positive predictive value for diagnosis of syphilis. In a screening setting, however, these tests give false-positive results at rates as high as 1-2%. Treponemal tests are likely to remain reactive even after adequate treatment and cannot differentiate past from current T. pallidum infection.

Treponemalimmunochromatographic strip (ICS) tests and enzyme immunoassays (EIAs), based largely on reactivity to recombinant antigens, have also been developed. Treponemal EIAs have been approved as confirmatory tests and, because of their ease of automation, are now used for screening purposes by some large laboratories. Because treponemal tests cannot distinguish between current and treated syphilis or may be falsely reactive, clinicians may be uncertain about how to interpret reactive EIA screening results.

Considerable interest has recently been focused on point-of-care ICS tests that can be used in the field or in resource-poor settings. These treponemal tests are not yet approved for use in the United States but have been assessed in antenatal clinics in a number of developing countries.

Source - Harrisons Principles of Internal Medicine, 18th Edition and e medicine articles

Gonorrhea

Gonorrhea is a sexually transmitted infection (STI) of epithelium and commonly manifests as cervicitis, urethritis, proctitis, and conjunctivitis. If untreated, infections at these sites can lead to local complications such as endometritis, salpingitis, tuboovarian abscess, bartholinitis, peritonitis, and perihepatitis in female patients; periurethritis and epididymitis in male patients; and ophthalmia neonatorum in newborns. Disseminated gonococcemia is an uncommon event whose manifestations include skin lesions, tenosynovitis, arthritis, and (in rare cases) endocarditis or meningitis.

Microbiology

Neisseria gonorrhoeae is a gram-negative, nonmotile, non-spore-forming organism that grows singly and in pairs (i.e., as monococci and diplococci, respectively). Exclusively a human pathogen, the gonococcus contains, on average, three genome copies per coccal unit; this polyploidy permits a high level of antigenic variation and the survival of the organism in its host. Gonococci, like all other Neisseria species, are oxidase positive. They are distinguished from other neisseriae by their ability to grow on selective media and to utilize glucose but not maltose, sucrose, or lactose.

Clinical Features

Gonococcal Infections in Males : Acute urethritis is the most common clinical manifestation of gonorrhea in males. The usual incubation period after exposure is 2-7 days, although the interval can be longer and some men remain asymptomatic. Urethral discharge and dysuria, usually without urinary frequency or urgency, are the major symptoms. The discharge initially is scant and mucoid but becomes profuse and purulent within a day or two. Gram's stain of the urethral discharge may reveal PMNs and gram-negative intracellular monococci and diplococcic. The clinical manifestations of gonococcal urethritis are usually more severe and overt than those of nongonococcal urethritis, including urethritis caused by Chlamydia trachomatis; however, exceptions are common, and it is often impossible to differentiate the causes of urethritis on clinical grounds alone.

Gonococcal Infections in Females

Gonococcal Cervicitis : Mucopurulent cervicitis is the most common. Cervicitis may coexist with candidal or trichomonal vaginitis. N. gonorrhoeae primarily infects the columnar epithelium of the cervical os. Bartholin's glands occasionally become infected.

Women infected with N. gonorrhoeae usually develop symptoms. However, the women who either remain asymptomatic or have only minor symptoms may delay in seeking medical attention. These minor symptoms may include scant vaginal discharge issuing from the inflamed cervix (without vaginitis or vaginosis per se) and dysuria (often without urgency or frequency) that may be associated with gonococcal urethritis. Although the incubation period of gonorrhea is less well defined in women than in men, symptoms usually develop within 10 days of infection and are more acute and intense than those of chlamydial cervicitis.

Physical Examination

The physical examination may reveal a mucopurulent discharge (mucopus) issuing from the cervical os. Because Gram's stain is not sensitive for the diagnosis of gonorrhea in women, specimens should be submitted for culture or a nonculture assay. Edematous and friable cervical ectopy as well as endocervical bleeding induced by gentle swabbing are more often seen in chlamydial infection. Gonococcal infection may extend deep enough to produce dyspareunia and lower abdominal or back pain. In such cases, it is imperative to consider a diagnosis of pelvic inflammatory disease (PID) and to administer treatment for that disease.

Gonococcal Vaginitis

The vaginal mucosa of healthy women is lined by stratified squamous epithelium and is rarely infected by N. gonorrhoeae. However, gonococcal vaginitis can occur in anestrogenic women (e.g., prepubertal girls and postmenopausal women), in whom the vaginal stratified squamous epithelium is often thinned down to the basilar layer, which can be infected by N. gonorrhoeae. The intense inflammation of the vagina makes the physical (speculum and bimanual) examination extremely painful. The vaginal mucosa is red and edematous, and an abundant purulent discharge is present. Infection in the urethra and in Skene's and Bartholin's glands often accompanies gonococcal vaginitis. Inflamed cervical erosion or abscesses in nabothian cysts may also occur. Coexisting cervicitis may result in pus in the cervical os.

Anorectal Gonorrhea

Because the female anatomy permits the spread of cervical exudate to the rectum, N. gonorrhoeae is sometimes recovered from the rectum of women with uncomplicated gonococcal cervicitis. The rectum is the sole site of infection in only 5% of women with gonorrhea. Such women are usually asymptomatic but occasionally have acute proctitis manifested by anorectal pain or pruritus, tenesmus, purulent rectal discharge, and rectal bleeding. Among men who have sex with men (MSM), the frequency of gonococcal infection, including rectal infection, fell by 90% throughout the United States in the early 1980s, but a resurgence of gonorrhea among MSM has been documented in several cities since the 1990s. Gonococcal isolates from the rectum of MSM tend to be more resistant to antimicrobial agents than are gonococcal isolates from other sites. Gonococcal isolates with a mutation in mtrR (multiple transferable resistance repressor) or in the promoter region of the gene that encodes for this transcriptional repressor develop increased resistance to antimicrobial hydrophobic agents such as bile acids and fatty acids in feces and thus are found with increased frequency in MSM. This situation may have been responsible for higher rates of failure of treatment for rectal gonorrhea with older regimens consisting of penicillin or tetracyclines.

Pharyngeal Gonorrhea

Pharyngeal gonorrhea is usually mild or asymptomatic, although symptomatic pharyngitis does occasionally occur with cervical lymphadenitis. The mode of acquisition is oral-genital sexual exposure, with fellatio being a more efficient means of transmission than cunnilingus. Most cases resolve spontaneously, and transmission from the pharynx to sexual contacts is rare. Pharyngeal infection almost always coexists with genital infection. Swabs from the pharynx should be plated directly onto gonococcal selective media. Pharyngeal colonization with Neisseria meningitidis needs to be differentiated from that with other Neisseria species.

Ocular Gonorrhea in Adults

Ocular gonorrhea in an adult usually results from autoinoculation from an infected genital site. As in genital infection, the manifestations range from severe to occasionally mild or asymptomatic disease. The variability in clinical manifestations may be attributable to differences in the ability of the infecting strain to elicit an inflammatory response. Infection may result in a markedly swollen eyelid, severe hyperemia and chemosis, and a profuse purulent discharge. The massively inflamed conjunctiva may be draped over the cornea and limbus. Lytic enzymes from the infiltrating PMNs occasionally cause corneal ulceration and rarely cause perforation.

Prompt recognition and treatment of this condition are of paramount importance. Gram's stain and culture of the purulent discharge establish the diagnosis. Genital cultures should also be performed.

Gonorrhea in Pregnant Women, Neonates, and Children

Gonorrhea in pregnancy can have serious consequences for both the mother and the infant. Recognition of gonorrhea early in pregnancy also identifies a population at risk for other STIs, particularly chlamydial infection, syphilis, and trichomoniasis. The risks of salpingitis and PID-conditions associated with a high rate of fetal loss-are highest during the first trimester. Pharyngeal infection, most often asymptomatic, may be more common during pregnancy because of altered sexual practices. Prolonged rupture of the membranes, premature delivery, chorioamnionitis, funisitis (infection of the umbilical cord stump), and sepsis in the infant (with N. gonorrhoeae detected in the newborn's gastric aspirate during delivery) are common complications of maternal gonococcal infection at term. Other microorganisms and conditions, including Mycoplasma hominis, Ureaplasma urealyticum, C. trachomatis, and bacterial vaginosis (often accompanied by infection with Trichomonas vaginalis), have been associated with similar complications.

The most common form of gonorrhea in neonates is ophthalmia neonatorum, which results from exposure to infected cervical secretions during parturition. Ocular neonatal instillation of a prophylactic agent (e.g., 1% silver nitrate eyedrops or ophthalmic preparations containing erythromycin or tetracycline) prevents ophthalmia neonatorum but is not effective for its treatment, which requires systemic antibiotics.

Clinical features

The clinical manifestations are acute and usually begin 2-5 days after birth. An initial nonspecific conjunctivitis with a serosanguineous discharge is followed by tense edema of the eyelids, chemosis, and a profuse, thick, purulent discharge. Corneal ulcerations that result in nebulae or perforation may lead to anterior synechiae, anterior staphyloma, panophthalmitis, and blindness. Infections described at other mucosal sites in infants, including vaginitis, rhinitis, and anorectal infection, are likely to be asymptomatic. Pharyngeal colonization has been demonstrated in 35% of infants with gonococcal ophthalmia, and coughing is the most prominent symptom in these cases. Septic arthritis is the most common manifestation of systemic infection or DGI in the newborn. The onset usually comes at 3-21 days of age, and polyarticular involvement is common. Sepsis, meningitis, and pneumonia are seen in rare instances.

Gonococcal Arthritis (DGI)

DGI (gonococcal arthritis) results from

gonococcal bacteremia. In the 1970s, DGI occurred in "0.5-3% of persons with untreated gonococcal mucosal infection. The lower incidence of DGI at present is probably attributable to a decline in the prevalence of particular strains that are likely to disseminate. DGI strains resist the bactericidal action of human serum and generally do not incite inflammation at genital sites, probably because of limited generation of chemotactic factors. Strains recovered from DGI cases in the 1970s were often of the PorB.1A serotype, were highly susceptible to penicillin, and had special growth requirements-including arginine, hypoxanthine, and uracil-that made the organism more fastidious and more difficult to isolate.

Menstruation is a risk factor for dissemination, and approximately two-thirds of cases of DGI are in women. In about half of affected women, symptoms of DGI begin within 7 days of onset of menses. Complement deficiencies, especially of the components involved in the assembly of the membrane attack complex (C5 through C9), predispose to neisserial bacteremia, and persons with more than one episode of DGI should be screened with an assay for total hemolytic complement activity.

Clinical features

The clinical manifestations of DGI have sometimes been classified into two stages: a bacteremic stage, which is less common today, and a joint-localized stage with suppurative arthritis. A clear-cut progression usually is not evident. Patients in the bacteremic stage have higher temperatures, and chills more frequently accompany their fever. Painful joints are common and often occur together with tenosynovitis and skin lesions. Polyarthralgias usually include the knees, elbows, and more distal joints; the axial skeleton is generally spared. Skin lesions are seen in "75% of patients and include papules and pustules, often with a hemorrhagic component. The differential diagnosis of the bacteremic stage of DGI includes reactive arthritis, acute rheumatoid arthritis, sarcoidosis, erythema nodosum, drug-induced arthritis, and viral infections (e.g., hepatitis B and acute HIV infection). The distribution of joint symptoms in reactive arthritis differs from that in DGI, as do the skin and genital manifestations.

Laboratory Diagnosis

A rapid diagnosis of gonococcal infection in men may be obtained by Gram's staining of urethral exudates. The detection of gram-negative intracellular monococci and diplococci is usually highly specific and sensitive in diagnosing gonococcal urethritis in symptomatic males but is only "50% sensitive in diagnosing gonococcal cervicitis. Samples should be collected with Dacron or rayon swabs. Part of the sample should be inoculated onto a plate of modified Thayer-Martin or other gonococcal selective medium for culture. It is important to process all samples immediately because gonococci do not tolerate drying. If plates cannot be incubated immediately, they can be held safely for several hours at room temperature in candle extinction jars prior to incubation. If processing is to occur within 6 h, transport of specimens may be facilitated by the use of nonnutritive swab transport systems such as Stuart or Amies medium. For longer holding periods (e.g., when specimens for culture are to be mailed), culture media with self-contained CO2-generating systems (such as the JEMBEC or Gono-Pak systems) may be used. Specimens should also be obtained for the diagnosis of chlamydial infection.

PMNs are often seen in the endocervix on a Gram's stain, and an abnormally increased number (30 PMNs per field in five 1000x oil-immersion microscopic fields) establishes the presence of an inflammatory discharge. Unfortunately, the presence or absence of gram-negative intracellular monococci or diplococci in cervical smears does not accurately predict which patients have gonorrhea, and the diagnosis in this setting should be made by culture or another suitable nonculture diagnostic method. The sensitivity of a single endocervical culture is "80-90%. If a history of rectal sex is elicited, a rectal wall swab (uncontaminated with feces) should be cultured. A presumptive diagnosis of gonorrhea cannot be made on the basis of gram-negative diplococci in smears from the pharynx, where other Neisseria species are components of the normal flora.

Increasingly, nucleic acid probe tests are being substituted for culture for the direct detection of N. gonorrhoeae in urogenital specimens. A common assay employs a nonisotopic chemiluminescent DNA probe that hybridizes specifically with gonococcal 16S ribosomal RNA; this assay is as sensitive as conventional culture techniques. A disadvantage of non-culture-based assays is that N. gonorrhoeae cannot be grown from the transport systems. Thus a culture-confirmatory test and formal antimicrobial susceptibility testing, if needed, cannot be performed. Nucleic acid amplification tests (NAATs), including Roche Amplicor, Gen-Probe APTIMA Combo2 (which also detects Chlamydia), and BD ProbeTec ET, offer an advantage: urine samples can be tested with a sensitivity similar to that obtained when urethral or cervical swab samples are assessed by culture and other non-NAATs.

Because of the legal implications, the preferred method for the diagnosis of gonococcal infection in children is a standardized culture. Two positive NAATs, each targeting a different nucleic acid sequence, may be substituted for culture of the cervix or the urethra as legal evidence of infection; however, cervical specimens are not recommended for prepubertal girls. Nonculture tests for gonococcal infection have not been approved by the U.S. Food and Drug Administration for use with specimens obtained from the pharynx and rectum of infected children. Cultures should be obtained from the pharynx and anus of both girls and boys, the vagina of girls, and the urethra of boys. For boys with a urethral discharge, a meatal specimen of the discharge is adequate for culture. Presumptive colonies of N. gonorrhoeae should be identified definitively by at least two independent methods.

Blood should be cultured in suspected cases of DGI. The use of Isolator blood culture tubes may enhance the yield. The probability of positive blood cultures decreases after 48 h of illness. Synovial fluid should be inoculated into blood culture broth medium and plated onto chocolate agar rather than selective medium because this fluid is not likely to be contaminated with commensal bacteria. Gonococci are infrequently recovered from early joint effusions containing <20,000 leukocytes/L but may be recovered from effusions containing >80,000 leukocytes/L. The organisms are seldom recovered from blood and synovial fluid of the same patient.

Differentiate between Syphilis and Gonorrhea

S.No.	Syphilis	Gonorrhea
Etiology	The *Treponema* species include *T. pallidum* subspecies *pallidum*, which causes venereal Syphilis.	*N gonorrhoeae* is a gram-negative, intracellular, aerobic diplococcus; more specifically, it is a form of diplococcus known as the gonococcus
Transmission and Epidemiology	Nearly all cases of syphilis are acquired by sexual contact with infectious lesions [i.e., the chancre, mucous patch, skin rash, or condylomatalata. Less common modes of transmission include nonsexual personal contact, infection in utero, blood transfusion, and organ transplantation.	*N gonorrhoeae* is spread by sexual contact or through vertical transmission during childbirth.It mainly affects the host's columnar or cuboidal epithelium. Virtually any mucous membrane can be infected by this microorganism. Gonococcal infection usually follows mucosal inoculation during vaginal, anal, or oral sexual contact. It also may be caused by inoculation of mucosa by contaminated fingers or other objects. Transmission through penile-rectal contact is fairly efficient.

Sex	Men are affected more frequently with primary or secondary syphilis than women.	The physiologic ectopy of the squamocolumnar junction onto the ectocervix in the adolescent female is one factor that causes particular susceptibility to this infection.
Prognosis	The morbidity of syphilis ranges from the relatively minor symptoms of the primary stages of infection to the more significant constitutional systemic symptoms of secondary syphilis and the significant neurological and cardiovascular consequences of tertiary disease. Since latent syphilis can persist for years or decades, the manifestations of tertiary syphilis often occur much later in life, causing significant morbidity and even death if not identified and treated.	With adequate early therapy, complete cure and return to normal function are the rule. Most gonococcal infections respond quickly to cephalosporin therapy. Late, delayed, or inappropriate therapy may lead to significant morbidity or, on rare occasions, death.
Clinical Presentation	Primary syphilis occurs 10-90 days after contact with an infected individual.	The incubation period for gonorrhea is usually 2-7 days after exposure to an infected partner. In all patients who present with a possible STD, the history should include the following:Past history of STDs (including HIV infection and viral hepatitis)Treatment history for known STDsKnown symptoms of STDs in current or past sexual partnersType of contraception usedAny history of sexual assault
Clinical Presentation	It manifests mainly on the glans penis in males and on the vulva or cervix in females. Ten percent of syphilitic lesions are found on the anus, fingers, oropharynx, tongue, nipples, or other extragenital sites. Regional nontender lymphadenopathy follows invasion.	The most common site of gonococcal infection in women is the endocervix (80%-90%), followed by the urethra (80%), rectum (40%), and pharynx (10%-20%). If symptoms develop, they often manifest within 10 days of infection. Major symptoms include vaginal discharge, dysuria, intermenstrual bleeding, dyspareunia (painful intercourse), and mild lower abdominal pain. In men, urethritis is the major manifestation of gonococcal infection. Initial characteristics include burning upon urination and a serous discharge. A few days later, the discharge usually becomes more profuse, purulent, and, at times, blood-tinged.
Clinical Presentation	Secondary syphilisSecondary syphilis manifests in various ways. It usually presents with a cutaneous eruption within 2-10 weeks after the primary chancre and	

	is most florid 3-4 months after infection. The eruption may be subtle; 25% of patients may be unaware of skin changes.	
Investigations	Serologic testing is considered the standard method of detection for all stages of syphilis.	Culture is the gold standard test in the workup of gonorrheal infections. Bacterial culture has high sensitivity and high specificity, is relatively low cost, is suitable for different types of specimen sources, and can be used for epidemiologic and resistance testing purposes. Perform a culture or nonculture detection test for *N gonorrhoeae* on endocervical, urethral, pharyngeal, or rectal discharge. Because organisms are intracellular, attempt to obtain specimens in a manner that will contain mucosal cells and not merely discharge (similar to a Papanicolaou smear).
Investigations	In suspected acquired syphilis, the traditional approach has been to first perform nontreponemal serology screening using the Venereal Disease Research Laboratory (VDRL), rapid plasma reagin (RPR), or the recently developed ICE Syphilis recombinant antigen test	Based on CDC guidelines and the USPS Task Force recommendations, nucleic acid amplification test (NAAT) assay is the test of choice to evaluate for urogenital infections in both males and females, whether symptomatic or asymptomatic
Serologic tests	Sensitivity of the VDRL and RPR tests are estimated to be 78-86% for detecting primary syphilis, 100% for detecting secondary syphilis, and 95-98% for detecting tertiary syphilis.	These tests include latex agglutination, ELISA, immunoprecipitation, and complement fixation tests. Because of their lower sensitivity and specificity, especially in populations with a low prevalence of disease, these tests are not routinely used for diagnosis, but they can be used as adjuncts to the other laboratory tests and may help in making the diagnosis.

MCQs

1. What is the most common type of gonococcal infection seen in newborns?
   a. Liver infection
   b. Ocular infection (conjunctivitis)
   c. Mastitis
   d. Meningitis
2. Which of the following disease is an STD
   a. Gonorrhea
   b. Syphilis
   c. Leprosy
   d. Both (a) and (b)
3. Gonorrhea is caused by which microorganism
   a. Neisseria gonorrhoeae
   b. Ventral pallidium
   c. Treponemapallidum
   d. C. perfringes
4. Causative microorganism in Syphilis
   a. Ventral pallidium
   b. Treponemapallidum
   c. Neisseria gonorrhoeae
   d. Mycoplasma genitalium

5. Incubation period of Primary syphilis occurs after how many days of contact with an infected individual

a. 10-90 days
b. 2-7 days
c. 7-14 days
d. 20-60 days

6. Chancre formation seen in

a. Gonorrhea
b. Leprosy
c. Syphilis
d. AIDS

Answer

1.	b	2.	d	3.	a
4.	b	5.	a	6.	c

CHAPTER

56

Krimi Roga

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

Introduction

Kṛmis or worms are of two varieties, namely, the *bāhya* (external) and *ābhyantara* (internal). They are further classified into four types as per their respective origin: 1. born out of external impurities, 2. generated by *kapha*, 3. generated by *Rakta* (blood) and 4. generated by faecal matter. These worms are 20 in number. The external ones are born out of impurities.

Persons taking regularly such food items that are raw, sweet and sour, large amount of water and watery beverages and the person who take incompatible eatables, jaggery; those who avoid physical exercise and those who sleep during day hours fall victims of *kṛmi roga*. A worm, insect; 'a spider'; a silk-worm; a shield-louse; an ant; lac (red dye caused by insects); an ant; lac (red dye caused by insects); Name of river (Used by *Mahābhārata* in this sense 6. 9. 17).

कृमि रोग हेतु एवं संप्राप्ति (Kṛmi Roga Hetu evam Samprāpti)

अजीर्णाध्यशनासात्म्यविरुद्धमलिनाशनैः ।
अव्यायामदिवास्वप्नगुर्वतिस्निग्धशीतलैः ॥
माषपिष्टान्नविदलबिसशालूकसेरुकैः ।
पर्णशाकसुराशुक्तदधिक्षीरगुडेक्षुभिः ॥
पललानूपपिशितपिण्याकपृथुकादिभिः ।
स्वाद्वम्लद्रवपानैश्च श्लेष्मा पित्तञ्च कुप्यति ।
कृमीम् बहुविधाकारान् करोति विविधाश्रयान् ॥

S.S.Utt. 54/3-6

Etiological factors and pathogenesis of *Kṛmi Roga* are as follows : Consumption of food which are uncooked, more in quantity, unaccustomed, incompatible and contaminated; not indulging in any physical exercises, sleeping during day time; food which is heavy, unctous and cold; those prepared from *Māsa*, cornflour, pulses, *Bisa*, *Śālūka* and *Kaseruka* & leafy vegetables; drinking *Surā*, *Śukta*, *Dadhi* & *Ksīra;* eating foods prepared from jaggery & sugarcane juice, consuming paste of *Tila*, meat of animals of marshy lands, *Piṇyāka* (paste of sesame added with jaggery), *Pṛthuka* (paddy dried & flattened), drinking beverages which are sweet & sour leading to aggravation of *Kapha* & *Pitta* and manifest *Kṛmi* of different shapes having different location.

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kapha* & *Pitta*
↓
Diminution of *Agni*
↓
Development of *Āma* inside the gastro intestinal tract
↓
Favours the genesis of *Kṛmi* of different shapes having different location.
↓
Kṛmi

कृमि रोग संप्राप्ति घटक (Samprāpti Ghaṭaka of Kṛmi Roga)

Doṣa : *Tridoṣaja (Kaphapradhāna)*
Dūṣya : *Dhātu* → *Rasa, Rakta, Māmsa, Meda, Asthi, Majjā*
Upadhātu → *Sirā, Tvacā, Snāyu*
Śārīrika Mala → *Puriṣa, Mūtra*
Dhātu Mala → *Kapha, Keśa, Loma, Nakha*
Agni : *Jaṭharāgni, Dhātvāgni, Bhūtāgni*
Agniduṣṭi : *Mandāgni*
Āma : *Āgnijanya*
Srotas : *Puriṣavaha, Annavaha, Raktavaha, Māmsavaha, Svedavaha*
Srotoduṣṭi : *Saṅga, Vimārgagamana*
Udbhavasthāna : *Āmāśayottha* & *Pakvāśayottha*

Adhiṣṭhāna	: *Āmapakvāśaya, Dhamani, Tvacā (Abhyantara Kṛmi); Tvacā (Bāhya Kṛmi)*
Sancārasthāna	: *Annavaha Srotoavayava, Dhamani, Tvacā*
Vyaktasthāna	: *Guda, Tvacā, Mukha*
Rogamārga	: *Abhyantara, Bāhya*
Svabhāva	: *Cirakāri & Āśukāri*
Prabhāva	: *Raktaja Kṛmi (Keśāda, Lomāda)*-incurable Newly manifested-curable
Roga bheda	: *Bāhya & Abhyantara*

कृमिभेद (Kṛmi Bheda)

विंशतेः कृमिजातीनां त्रिविधः सम्भवः स्मृतः ।
पुरीषकफरक्तानि तासां वक्ष्यामि विस्तरम् ॥
S.S.Utt. 54/7

अथास्मै प्रोवाच भगवानात्रेयः–इह खल्वग्निवेश ! विंशतिविधाः क्रिमियः पूर्वमुद्दिष्टा नानाविधेन प्रविभागेनान्यत्र सहजेभ्यः; ते पुनः प्रकृतिभिर्विभज्यमानाश्चतुर्विधा भवन्ति; तद्यथा–पुरीषजाः, श्लेष्मजा, शोणितजा, मलजाश्चेति ॥
C.S.Vi. 7/9

There are twenty varieties of *Kṛmi* are described excluding the normal ones which are already mentioned. Based on their source, they are again classified into four groups namely *Puriṣaja, Śleṣmaja, Śoṇitaja* and *Malaja.*

कृमिरोग सामान्य लक्षण (Kṛmi Roga Sāmānya Lakṣaṇa)

ज्वरो विवर्णता शूलं हृद्रोगः सदनं भ्रमः ।
भक्तद्वेषोऽतिसारश्च सञ्जातकृमिलक्षणम् ॥
S.S.Utt. 54/18-19

General signs & symptoms of *Kṛmiroga* are as follows : Fever, discolouration of the body, pain in the abdomen, heart disease, general malaise, giddiness, aversion to food and diarrhoea.

मलज कृमि (Malaja Kṛmi)

तत्र मलो बाह्यश्चाभ्यन्तरश्च । तत्र बाह्यमलजातान् मलजान् संचक्ष्महे । तेषां समुत्थानंमृजावर्जनं; स्थानं–केशश्मश्रुलोमपक्ष्मवासांसि; संस्थानम्अणवस्तिलाकृतयो बहुपादाश्च; वर्णः–कृष्णः, शुक्लश्च; नामानि-यूकाः, पिपीलिकाश्च; प्रभावः–कण्डूजननं, कोठपिडकाभिनिर्वर्तनं च; चिकित्सितं तु खल्वेषामपकर्षणं, मलोपघातः, मलकराणां च भावानामनुपसेवनमिति ॥
C.S.Vi. 7/10

Two varieties of excreta described namely external as well as internal. External varieties of *Kṛmi* are described under *Malaja Kṛmi.*

Etiology	: Avoidance of cleanliness.
Habitat	: Hairs, beards, mustaches, small hairs, eyelashes & clothing.
Form	: Minute, sesamum shaped & multipede.
Colour	: Black & white
Name	: *Yūkā, Pipīlikā*
Clinical features	: Itching, urticarial patches and pimples.
Treatment	: Comprises of extraction, removal of excreta and avoidance of factors which produce the excreta.

रक्तज कृमि (Raktaja Kṛmi)

शोणितजानां तु खलु कुष्ठैः समानं समुत्थानं; स्थानं-रक्तवाहिन्यो धमन्यः; संस्थानम्-अणवो वृत्ताश्चापादाश्च, सूक्ष्मत्वाच्चैके भवन्त्यदृश्याः; वर्णः–ताम्रः; नामानि-केशादा, लोमादा, लोमद्वीपाः, सौरसा, औडुम्बरा, जन्तुमातरश्चेति; प्रभावः–केशश्मश्रुनखलोमपक्ष्मापध्वंसः, व्रणगतानां च हर्ष-कण्डूतोदसंसर्पणानि, अतिवृद्धानां च त्वक्सिरास्नायुमांसतरुणास्थिभक्षणमिति; चिकित्सितमप्येषां कुष्ठैः समानं, तदुत्तरकालमुपदेक्ष्यामः ॥
C.S.Vi. 7/11

केशरोमनखादाश्च दन्तादाः किक्किशास्तथा ।
कुष्ठजाः सपरीसर्पा ज्ञेयाः शोणितसम्भवाः ॥
ते सरक्ताश्च कृष्णाश्च स्निग्धाश्च पृथवस्तथा ।
रक्ताधिष्ठानजान् प्रायोविकारान् जनयन्ति ते ॥
S.S.Utt. 54/15-16

विरुद्धाजीर्णशाकाद्यैः शोणितोत्था भवन्ति हि ॥
S.S.Utt. 54/18

Etiology	: Similar to that of *Kuṣṭha.* Ingestion of incompatible foods, uncooked vegetables.
Habitat	: *Raktavāhi Dhamani*
Form	: Minute, round, having no pedicle, some of them are invisible, unctous and thick in size.
Colour	: Coppery, red or black
Names	: *Keśāda, Lomāda, Lomadvīpa,*

Saurasā, Audumbarā and *Jantumātara (Caraka). Kesāda, Romāda, Nakhāda, Dantāda, Kikkisa, Kuṣṭhaja & Parīsarpa (Suśruta).*

Clinical features : Falling of hairs, beard, mustaches, nails, small hairs, eyelashes.

Hyperasthesia, itching, pain & creeping sensation in the ulcerated area when excessive grown, they eat away skin, vessels, ligaments, muscle tissue and cartilages and manifest diseases affecting the seats of blood.

Treatment : Like *Kuṣṭha.*

श्लेष्मज कृमि (Śleṣmaja Kṛmi)

मांसमाषगुडक्षीरदधितैलैः कफोद्भवाः ॥

S.S.Utt. 54/17

श्लेष्मजाः क्षीरगुडतिलमत्स्यानूपमांसपिष्टान्नपरमान्नकुसुम्भस्नेहाजीर्णपूतिक्लिन्नसंकीर्णविरुद्धासात्म्यभोजनसमुत्थानाः; तेषामामाशयः स्थानं, ते प्रवर्धमानास्तूर्ध्वमधो वा विसर्पन्त्युभयतो वा; संस्थानवर्णविशेषास्तु–श्वेताः पृथुब्रध्नसंस्थानाः केचित्, केचिद्वृत्तपरिणाहा गण्डूपदाकृतयः श्वेतास्ताम्रावभासाश्च, केचिदणवो दीर्घास्तन्त्वाकृतयः श्वेताः; तेषां त्रिविधानां श्लेष्मनिमित्तानां क्रिमीणां नामानि-अन्त्रादाः, उदरादाः, हृदयचराः, चुरवः, दर्भपुष्पाः, सौगन्धिकाः, महागुदाश्चेति; प्रभावोहृल्लासः, आस्यसंस्रवणम्, अरोचकाविपाकौ, ज्वरः, मूर्च्छा, जृम्भा, क्षवथुः, आनाहः, अङ्गमर्दः, छर्दिः कार्श्यं, पारुष्यं, चेति ॥

C.S.Vi. 7/12

दर्भपुष्पा महापुष्पाः प्रलूनाश्चिपिटास्तथा।
पिपीलिका दारुणाश्च कफकोपसमुद्भवाः ॥
रोमशा रोममूर्द्धानः सपुच्छाः श्यावमण्डलाः।
रूढधान्याङ्कुराकाराः शुक्लास्ते तनवस्तथा ॥
मज्जादा नेत्रलेढारस्तालुश्रोत्रभुजस्तथा।
शिरोहृद्रोगवमथुप्रतिश्यायकराश्च ते ॥

S.S.Utt. 54/12-14

Etiology : Consumption of food consisting of milk, jaggery, sesamum, fish, meat of marshy animals, flour preparations, rice cooked in milk, oil of *Kusumbha;* food which is uncooked, putrified, stale, infected, antagonistic and unsuitable items, ingestion of meat, *Māsa,* curd & oil.

Habitat : *Āmāśaya,* when excessivelly grown, they move upwards, or to both the sides.

Form and colour : Some are broad tape like and white in colour. Some are round like earthworms, white & coppery colour. Some are minute, long and thread like and white in colour. These have hairs on the head and body, blue patches on their body, have tail, resemble sprouted grain in shape, white & thin.

Names : *Antrāda, Udarāda, Hṛdayacara, Curu, Darbhapuṣpa, Saugandhika, Mahāguda (Caraka) Darbhapuṣpa, Mahāpuṣpa, Pralūna, Cipiṭa, Pipīlikā* and *Dāruṇa (Suśruta)*

Clinical features : Nausea, salivation, anorexia, indigestion, fever, fainting, yawning, sneezing, constipation, malaise, vomiting, emaciation & dryness of the body. These consume bonemarrow, affect the eyes, palate, ears & shoulders. Diseases of head, heart, *pratiśyāya* etc.

पुरीषज कृमि (Purīṣaja Kṛmi)

पुरीषजास्तुल्यसमुत्थानाः श्लेष्मजैः; तेषां स्थानं पक्वाशयः, ते प्रवर्धमानास्त्वधो विसर्पन्ति, यस्य पुनरामाशयाभिमुखाः स्युर्यदन्तरं तदन्तरं तस्योद्गारनिःश्वासाः पुरीषगन्धिनः स्युः; संस्थानवर्णविशेषास्तुसूक्ष्मवृत्तपरीणाहाः श्वेता दीर्घा ऊर्णांशुसंकाशाः केचित्, केचित् पुनः स्थूलवृत्तपरीणाहाः श्यावनीलहरितपीताः; तेषां नामानि ककेरुकाः, मकेरुकाः, लेलिहाः; सशूलकाः, सौसुरादाश्चेति; प्रभावः–पुरीषभेदः, कार्श्यं, पारुष्यं, लोमहर्षाभिनिर्वर्तनं च, त एव चास्य गुदमुखं परितुदन्तः कण्डूं चोपजनयन्तो गुदमुखं पर्यासते, त एव जातहर्षागुदनिष्क्रमणमतिवेलं कुर्वन्ति; इत्येय श्लेष्मजानां पुरीषजानां च क्रिमीणां समुत्थानादिविशेषः ॥ *C.S.Vi. 7/13*

माषपिष्टान्नविदलपर्णशाकैः पुरीषजाः।

S.S.Utt. 54/16

अजवा विजवाः किप्याश्चिप्या गण्डूपदास्तथा ।
चुरवो द्विमुखाश्चैव ज्ञेयाः सप्त पुरीषजाः ॥
श्वेताः सूक्ष्मास्तुदन्त्येते गुदं प्रतिसरन्ति च ।
तेषामेवापरे पुच्छैः पृथवश्च भवन्ति हि ॥
शूलाग्निमान्द्यपाण्डुत्वविष्टम्भबलसङ्क्षयाः ।
प्रसेकारुचिहृद्रोगविड्भेदास्तु पुरीषजैः ॥
रक्ता गण्डूपदा दीर्घा गुदकण्डूनिपातिनः ।
शूलाटोपशकृद् भेदपक्तिनाशकराश्च ते ॥

S.S.Utt. 54/8-11

Etiology : All the etiological factors mentioned under *Śleṣmaja Kṛmi*. Intake of foods prepared from *Māṣa, Piṣṭānna*, pulses & leafy vegetables.

Habitat : *Pakvāśaya*, when developed they move downwards; when they move towards *Āmāśaya*, then the eructation and breath of the patient produce fecal odour.

Form & Colour : Some are minute, cylindrical, white, long, like wool fibers; some are gross, cylindrical, blackish, blue, green and yellow. These are white in colour, small, irritate the anal region, come out of the anus, among these some are big in their tails portion.

Names : *Kakeruka, Makeruka, Lelihā, Saśūlakā* and *Sausurāda (Caraka) Ajava, Vijava, Kipyā, Cipya, Gaṇḍūpada, Curava* and *Dvimukha (Suśruta)*

Clinical features : Diarrhoea, emaciation, dryness, horripilation, when they reach anus, they produce piercing pain & itching in anal region; when they excited they frequently come out of the anus, pain in the abdomen, dyspepsia, anaemia, constipation, loss of strength, nausea, loss of taste, heart disease. *Gaṇḍūpada Krimi* are red in colour and long, produces itching of the anus, abdominal pain, gurgling sound in the abdomen, diarrhoea & loss of digestive power.

साध्यसाध्यता (Sādhyāsādhyatā)

दृश्यास्त्रयोदशाद्यास्तु कृमीणां परिकीर्तिताः ।
केशादाद्यास्त्वदृश्यास्ते द्वावाद्यौ परिवर्जयेत् ॥

S.S.Utt. 54/19-20

Thirteen varieties i.e. *Puriṣaja* & *Kaphaja* are visible seven varieties of *Keśāda* etc. are invisible.

Keśāda & *Romāda* should be rejected for treatment.

उपशय/पथ्य (Upaśaya/Pathya)

सुरसादिन्तु सर्वेषु सर्वथैवोपयोजयेत् ॥
प्रव्यक्ततिक्तकटुकं भोजनञ्च हितं भवेत् ।
कुलत्थक्षारसंसृष्टं क्षारपानश्च पूजितम् ॥
क्षीराणि मांसानि घृतानि चैव दधीनि शाकानि च पर्णवन्ति ।
समासतोऽम्लान्मधुरान् हिमांश्च कृमीन् जिघांसुः परिवर्जयेत्तु ॥

S. S.Utt. 54/38-40

Surasādi gaṇa drugs of all forms are benefecial for the management of all varieties of *Kṛmi.*

Food which are predominant in bitter and pungent taste will be beneficial. Drinking *Yavakṣāra* mixed with ash of *Kulattha* is highly claimed as beneficial.

The person desirous of getting rid off *Kṛmi* should avoid use of milk, meat, ghee, curd, leafy vegetables, those which are sour, sweet and cold substances.

Application of *Āsthāpana Basti*, *Virecana*, *Śirovirecana*, smoke treatment, bitter, sour and hot eatables that controls *Kapha* contents, massage that revitalises the body, *Utsādana*, rice of red *Śāli* variety, leaves of *Paṭola*, leaves of the shoots of bamboo, garlic pearls and leaves of *Vāstuka*, all these forms a very useful diet. (B.R. 11/83)

Vegetable leaves of *Citraka*, *Arka* plant, mustard plant, vegetable of unripe banana fruits, bitter vegetables, vegetable leaves of *Nāḍīca*, meat of mouse, vegetable leaves of *Vāyaviḍaṅgam* and *Neem* are useful diets for those indisposed due to worm infestation. (B.R. 11/84)

Haraḍa, oils of sesame and mustard, *Sauvīra*, *Śukta*, *Tuṣodaka*, honey, ripe palm fruits, purified *Bhallātaka*, cow's urine, betel leaves, liquor and *Kastūrī* are very useful. (B.R. 11/85)

Urine of she-camel, *Ghṛta* and milk, asafoetida fried in *Ghṛta*, *Yavakṣāra*, *Ajamodā*, *Khadira*, *Indrayava*, juice of *Jambūra* lemon, vegetable leaves of *Kāravellaka*, caraway seeds, *Agara*, *Śimśapa* and its abstracts are also beneficial. (B.R. 11/86)

The intake of sour, bitter and pungent juices are beneficial for the patients suffering from worm diseases. One should take daily the eatables that have sour, bitter and pungent taste and the meals that control *Kapha*. The stomachiac and tasteful meals are also suggested. (B.R. 11/87)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

Avoidable meals in cases of *Kṛmi*: One should avoid meals and medicines that cause vomiting. However, one should not hold the vomitting if appearing on its own. Incompatible meals like fish and milk taken together should be avoided. Like wise one should not sleep in daytime, excessive intake of liquid *Kalka* based meals are also forbidden. Pulse beans of *Urada*, curd, spinach, meat, milk of animals other than camel and sweet items are also prohibited. (B.R. 11/89)

MCQs

1. *Churava Krumi* is
   a. *Raktaja* b. *Kaphaja*
   c. *Purishaja* d. *Bahya*
2. *Kakeruka Krimi* is
   a. *Raktaja* b. *Kaphaja*
   c. *Purishaja* d. *Bahya*
3. *Krimi is mentioned as a Samkramikaroga by which Acharya*
   a. *Caraka* b. *Susruta*
   c. *Bhavaprakasa* d. *Yogaratnakara*
4. Number of types of *Krimi* as per *Susrutain* the order of *Purishaja, Raktaja* and *Kaphaja Krimi*
   a. 5,6,7 b. 6,7,6
   c. 6,7,7 d. 7,7,6
5. Which *Krimi* is invisible
   a. *Raktaja* b. *Kaphaja*
   c. *Purishaja* d. *Bahya*
6. Types of *Krimi* as per *Sharangadhara*
   a. 2 b. 3
   c. 21 d. 20
7. '*Virudhabhojana*' is the *Nidana* of
   a. *Raktaja* b. *Kaphaja*
   c. *Purishaja* d. *Bahya*
8. According to *Sushruta*, *Pandutva* is caused by
   a. *Raktaja* b. *Kaphaja*
   c. *Purishaja* d. *Bahya*
9. *Asraya* of *Purishaja Krimi* as per *Susruta*
   a. *Amasaya* b. *Pakwasaya*
   c. *Koshta* d. *Guda*
10. *Tilapramana* is the feature of which type of *Krimi*
   a. *Bahya* b. *Raktaja*
   c. *Kaphaja* d. *Pureeshaja*
11. Which of the following is not considered as the feature of *Raktajakrimi*
   a. *Bahupada*
   b. *Apada*
   c. Adarshana
   d. *Raktavahisirothanam*

Answer

1.	b	2.	c	3.	b
4.	d	5.	a	6.	c
7.	a	8.	c	9.	a
10.	a	11.	a		

CHAPTER

57

Clinical Presentation of Common Parasitic Disorders

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

Medical Technology

Medical Technology is indispensable to healthcare delivery. It is an integral part of the entire healthcare lifecycle, from the stage of screening/ diagnosing to treatment/care; from restoring patients to normal lives and monitoring their health post treatment. For decades now, with advancement of technology and increasing sophistication of medical devices, the industry has played a major role in bringing down the incidence of disease and improving the overall healthcare system across the world. Globally, the healthcare sector is witnessing rapid advancements in how patients are diagnosed and treated and the Medical Technology industry is at the core of this transformation. The industry is well positioned to revolutionize healthcare in India as well. Using a mix of nascent as well as advanced technologies such as artificial intelligence, remote sensing, 3D printing, e-ICUs, virtual reality, telemedicine and others has the potential to help India's healthcare industry overcome many of the challenges faced by it- be it accessibility.

1. Affordability or quality Health care technology is capable of providing cost -effective solutions at a time when the demands on health
2. Social care services continue to increase. At the moment, the pace of change in the technology is increasing at an exponential rate.
3. Rapid technological change will require retraining and rethinking the roles of clinicians. Both existing and future generations of clinicians will need training in digital literacy to be able to make use of the 3 technology in providing optimum care. The future of healthcare lies in working hand-in-hand with technology
4. Healthcare workers have to embrace emerging healthcare technologies in order to stay relevant in the coming years
5. Taking all this into consideration, Public Health Foundation of India (PHFI), Association of Health Providers India (AHPI), Indian Institute of Science (IISc), Bangalore and Indian Institute of Space Science and Technology (IIST) have collaborated for a Certificate course on Healthcare Technology which will combine theoretical and practical aspects of healthcare technology at every level of healthcare delivery and personnel involved as per requirement.

Hookworm

- Human hookworm disease is a common helminth infection worldwide that is predominantly caused by the nematode parasites *Necator americanus* and *Ancylostoma duodenale*; organisms that play a lesser role include *Ancylostoma ceylanicum, Ancylostoma braziliense*, and *Ancylostoma caninum.*
- Hookworm infection is acquired through skin exposure to larvae in soil contaminated by human feces.
- Soil becomes infectious around 5-10 days after contamination and remains so for 3-4 weeks, depending on conditions

Clinical Presentation

History

- The majority of individuals who develop hookworm infection are from known endemic areas. They frequently have a history of wearing open footwear or walking barefoot in such areas.
- Most individuals with hookworm infection are asymptomatic, and diagnosis is made only by means of stool examination.
- Those symptoms that do occur depend on the type of hookworm disease present (ie, classic hookworm disease, cutaneous larva migrans,

or eosinophilic enteritis) and on the stage of the disease (ie, early or late).

Early symptoms of classic hookworm disease

- During the first 1-2 weeks after a cutaneous infection, hookworm produces a local irritation at the site of infection, termed ground itch or dew itch.
- An intensely pruritic, erythematous, or vesicular rash appears, usually on the feet or hands.
- Cough and wheezing can occur about 1 week after exposure as a consequence of larval migration through the lungs.
- Pulmonary symptoms are uncommon and usually mild, except in severe infections.
- In rare cases, severe infections may give rise to Liffler syndrome, characterized by paroxysmal attacks of cough, dyspnea, pleurisy, little or no fever, and eosinophilic pulmonary infiltrates that last several weeks after the initial infection.
- Migration of the worms into the gastrointestinal (GI) tract may cause GI discomfort secondary to irritation. As the worms mature in the jejunum, patients may experience diarrhea, vague abdominal pain, colic, flatulence, nausea, or anorexia. These symptoms are more common with initial exposures than with subsequent exposure and typically peak between 30 and 45 days after infection.
- In people who have been infected with a large burden of *A duodenale* through oral ingestion, Wakana syndrome may occur. This syndrome resembles an immediate-type hypersensitivity reaction and is characterized by pharyngeal itching, hoarseness, nausea, vomiting, cough, dyspnea, and eosinophilia.

Later symptoms of classic hookworm disease

- Mild hookworm infections are often asymptomatic.
- Moderate-to-heavy infections cause significant blood loss, which may manifest as melena.
- Once iron reserves are exhausted, anemia develops.
- A large worm burden and a history of poor iron intake increase the likelihood of significant anemia.
- Patients with severe iron-deficiency anemia may present with lassitude, headache, palpitations, exertional dyspnea, syncope, or edema. They may also have a history of perverted taste and pica. In rare cases, anemia may provoke ischemic symptoms such as angina or claudication.
- Deficits in physical and intellectual growth can occur due to anemia and protein deficiency; these deficits may be irreversible when they develop during childhood.

Cutaneous larva migrans

Infection with zoonotic hookworms, especially *A. braziliense*, can progress with a lateral skin migration of larvae those results in the characteristic tracts of cutaneous larva migrans (creeping eruption). This is to be distinguished from the ground itch noted in classic hookworm disease.

Eosinophilic enteritis

- Eosinophilic enteritis is characterized by repeated episodes of abdominal pain in approximately 97% of affected individuals. These episodes typically occur with increasing severity and are associated with peripheral eosinophilia in almost 100% of patients and with leukocytosis in approximately 75% of patients.
- Extreme cases may mimic appendicitis or intestinal perforation.

Physical Examination

Skin and pulmonary findings are minimal. Physical findings in the early (larval migration) stage of the disease differ from those in the late (established GI infection) stage.

Early signs of classic hookworm disease

- An erythematous, pruritic, papulovesicular rash develops at the site of initial infection and may persist for 1-2 weeks after initial infection.
- Intense scratching may lead to a secondary bacterial infection, which is quite common.

- When the worms break through from the venous circulation into the pulmonary air spaces, cough, fever, and a reactive bronchoconstriction may be observed, with wheezing heard on auscultation.
- During the period of intestinal involvement, abdominal examination may reveal midepigastric pain on palpation.
- Stools may be bloody or melanotic.

Later signs of classic hookworm disease

- Signs of iron-deficiency anemia are often insensitive.
- Patients may exhibit pallor, chlorosis (greenish-yellow skin discoloration), hypothermia, spooning nails, tachycardia, or signs of high-output cardiac failure.
- Hypoproteinemia may lead to anasarca and peripheral edema.
- Poor skin texture, edema, and susceptibility to cutaneous infection suggest possible malnutrition.
- Stunted growth may be observed in children with severe infection.

Cutaneous larva migrans

- Cutaneous larva migrans manifests as pathognomonic, raised serpiginous tracts (creeping eruptions) with surrounding erythema that may last as long as 1 month if untreated.
- Lesions are most commonly seen on lower extremities but may be limited to the trunk or upper extremities, depending on the site at which the infective larvae entered the body.

Ancillary investigations –workup

Complete blood count [CBC])

- It may detect iron-deficiency anemia.
- A differential count may reveal eosinophilia (1000-4000 cells/L).

Stool for ova and parasites

- Stool should be examined for ova and parasites.
- The examination may demonstrate significant number of hookworm eggs. However, because egg laying may be delayed, stool examination should not be considered a sensitive test for identifying hookworm infection.
- Stool examinations may have to be repeated.

PCR-based methods

- PCR-based methods for the sensitive and specific diagnosis of hookworm have been developed, but are not widely used.
- Other causes of iron loss and blood loss should be excluded.

Other investigations

- In cases of severe infection, chest radiography may show diffuse alveolar infiltrates during the migration of the larvae through the lung to the intestine.
- Once infection is established in the gut, imaging studies are not helpful.
- In cases of eosinophilic enteritis, colonoscopy may reveal ileal and colonic ulceration and, occasionally, adult hookworms.

Ascariasis/Roundworms

- Ascariasis is the most common helminthic infection, with an estimated worldwide prevalence of 804 million cases in 2013.
- It is caused by *Ascaris lumbricoides*, which is the largest parasite from the class of nematodes commonly known as roundworms.
- Usually *A lumbricoides* or *A suum* infection is asymptomatic.
- Ascariasis is most prevalent in children of tropical and developing countries, where they are perpetuated by contamination of soil by human feces or use of untreated feces as fertilizer and the water or food contaminated by the eggs is ingested.
- Symptomatic ascariasis occurs during the adult worm's intestinal or larval migration phase and may manifest as growth retardation, pneumonitis, intestinal obstruction, or hepatobiliary and pancreatic injury.

- Ascariasis may exist as a zoonotic infection associated with pigs and use of hog manure and is caused by *Ascaris suum*, which is very similar to *Ascaris lumbricoides* but, in most endemic areas, it is most likely transmitted from person to person.

Clinical presentation

History

- Early symptoms of ascariasis, during the initial lung migration (first 10-14 days after ingestion) include cough, dyspnea, wheezing, urticaria, hemoptysis, and chest pain.
- This may be seasonal after rains in some countries, such as Saudi Arabia. Returning travelers could also present with Loeffler syndrome of eosinophilic pneumonia if exposed in a high-risk environment.
- Abdominal pain or discomfort, distension, colic, nausea, anorexia, and intermittent diarrhea may be manifestations of partial or complete intestinal obstruction by adult worms during the late phase of infection six to eight weeks after egg ingestion.
- Adult worms are passed in the stool and can be seen on stool examination.
- Jaundice, nausea, vomiting, fever, and severe or radiating abdominal pain may suggest cholangitis, pancreatitis, hepatic abscess, or appendicitis.

Physical

- Within 1 to 2 weeks of new infection following egg ingestion, *Ascaris* larvae migrate through lungs causing eosinophilic pneumonitis. This is known as the Loeffler syndrome.
- Parasitic infections with *Strongyloides stercolis*, *Ancylostoma duodenale*, *Necator americanus, Toxocara*, schistosomiasis, and lymphatic filariasis can present with a similar pathological presentation.
- Patients may present with fever, dry cough, dyspnea, wheezing, and urticaria that subsides within 5 to 10 days.
- Rales, wheezes, and tachypnea may develop during pulmonary migration, particularly in persons with a high worm burden.
- Urticaria and fever may also develop late in the migratory phase.
- Abdominal distension is nonspecific, but is common in children with ascariasis.
- Abdominal tenderness, especially in the right upper quadrant, hypogastrium, or right lower quadrant, may suggest complications of ascariasis.
- Evidence for nutritional deficiency due to ascariasis is strongest for vitamins A and C, as well as for protein, as indicated by albumin and growth studies in children observed prospectively.

Ancillary investigations–workup

Stool examination for ova and parasites:

- Visualization of large, brown 60 m̄ X 50 m̄ trilayered eggs is confirmatory.
- However, stool examination may be negative for ova for up to 40 days after infection because of the time needed for migration and maturation of the worm.
- However, it may take 40 days for eggs to appear in the stools and if only infested with male worms, there may be no eggs at all.

Sputum examination: *Ascaris* larvae may be observed in microscopic wet preparations of sputum during the pulmonary migration phase.

CBC with differential: Results will reveal peripheral eosinophilia during the tissue migration phase of the infection. It may take weeks to resolve, but can be masked with the administration of steroids. It is more likely in the early lung phase than the late phase.

Serological tests: Serum IgG and IgE are elevated during early infection, but this finding is not clinically useful.

Pinworm (Enterobiasis)/ Threadworms

- Enterobius (syn. Oxyuris) vermicularis is a human-pathogenic intestinal parasite belonging to the nematodes (Nematoda).
- Synonyms include "threadworm" and "seatworm."

- Symptomatic pinworm infection is referred to as enterobiasis (older term: oxyuriasis).
- Threadworms, also called pinworms, are tiny parasites that grow to about 1cm in length and look like tiny pieces of white thread. They live in the intestines.
- In humans, they reside in the cecum, appendix, and ascending colon.
- Female pinworms are 8-13 mm long, and males are 2-5 mm long.
- Pinworm infection is primarily a pediatric condition, and parents are typically infected via transmission through their children.
- Pinworm is prevalent throughout the temperate regions of the world and is the most common helminthic infection in the United States.
- Transmission can occur via direct contact with contaminated furniture, bedclothes, bedding, towels, toilets, doorknobs, or other objects. The parasite also can be transmitted during sexual contact.
- Pinworm infection generally is asymptomatic; asymptomatic carriers are common.
- The cure rate with treatment is 90-95%. Re-infection is common, especially if all contacts are not treated simultaneously.

Clinical presentation and diagnosis

History and Physical Examination

- The patient history of pinworm infection, also called enterobiasis, includes itching or prickling pain in the anal area (usually at nighttime or in the early morning), intense anal itching, restless sleep or difficulty sleeping, and, rarely, abdominal discomfort or loss of appetite.
- Other symptoms include irritability and, in females, vaginal itching. Most patients, however, are asymptomatic.
- The pale-colored female pinworm (10 mm) may be seen in the perianal region with the naked eye. The worm's appearance is easily confused with bits of cotton thread.
- Eggs (30 m̄ X 50-60 m̄) usually are not seen without a microscope.
- Perianal excoriations secondary to scratching may be found.

Ancillary investigations and workup

Stool for ova and parasites

- Glass slide microscopic analysis for Enterobius vermicularis may be performed looking for ova and female pinworms.
- A specimen is best obtained by dabbing the stretched, unwashed perianal folds in the early morning with cellophane tape and affixing the specimen onto a slide. A negative test for 5 consecutive mornings effectively rules out the diagnosis.

Note-

- Stool microscopy is not a helpful diagnostic tool, since the worm eggs are deposited outside the intestine.
- Serological methods are of no diagnostic relevance.
- Similarly, neither blood eosinophilia nor elevated immunoglobulin E levels are generally expected due to the low invasiveness of the worms.

MCQs

1. Diagnosis of Enterobiusvermicularis (pinworm) is done by
   a. Perianal skin
   b. Feces
   c. Sputum
   d. Duodenal fluid
2. Which of the following is the most common symptom of pinworm (Enterobiusvermicularis) infection?
   a. Ulcerative lesions in the small or large bowel
   b. Abdominal distention and cramps
   c. Nocturnal perianal pruritus
   d. Vomiting and watery diarrhea
3. The causative organism for roundworm infestation
   a. Ascarislumbricoides
   b. Enterobiusvermicularis
   c. Ancylostomaduodenale
   d. Necatoramericanus

4. Loeffler syndrome seen in
   a. Aspergillosis
   b. Coccidioidomycosis
   c. Ascariasis
   d. Schistosomiasis
5. The stool examination for Ascariasis is positive after how many days
   a. More than 1 week
   b. More than 40 days
   c. More than 14 days
   d. More than 20 days
6. Wakana syndromeseen in
   a. Oral ingestion of Ancylostomaduodenale
   b. Cutaneous infection of Ancylostoma-duodenale
   c. Oral ingestion of Enterobiusvermicularis
   d. Cutaneous infection of Enterobiusve-rmicularis

Answer

1.	a	2.	c	3.	a
4.	c	5.	b	6.	a

CHAPTER

58

Khalitya and Palitya

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

खालित्य (Khālitya)

तेजोनिलौः सह केशभूमिं दग्ध्वाऽऽशु कुर्यात् खलतिं नरस्य ।
C.S.Ci. 26/132-133

Baldness of head due to loss of scalp hair is known as *Khālitya* or *Khalita* and it is a disease of scalp. It is caused by vitiation and aggravation of *pitta* and *vāta doṣa* together and cause *dagda* or burning of scalp (*keśa bhūmi*) leading to the hair fall due to morbidity in the hair follicles.

रोमकूपानुगं पित्त वातेन सह मूर्च्छितम् ।
प्रच्यावयति रोमाणि ततः श्लेष्मा सशोणितः ॥
रुणद्धि रोमकूपांस्तु ततोऽन्येषामसंभवः ।
तदिन्द्रलुप्तं खालित्यं रुज्येति च विभज्यते ॥
S.S.Ni. 13/33-34

Exacerbated and vitiated *Pitta* and *vāta* reaches *romakūpa* (pore of the skin/hair follicle) and burns the scalp skin and demolish *romakūpa* leading to hair fall. Intensified *Kapha* and shonita causes thickening of the scalp due to their impaired the qualities resulting into the blockage of hair follicles and causes no more new hair growth.

Indralupta

The falling of the scalp hair is also known as *Indralupta*. The difference between *Indralupta* and *Khālitya*

Indralupta	Khālitya
Patchy hair loss in the scalp	Huge area of scalp is involved
Loss of hairs is sudden	Loss of hairs is gradual

वातज खालित्य (*Vātaja Khālitya*)

The Skin of the scalp turns dry due to properties of *vāta*.

पित्तज खालित्य (*Pittaja Khālitya*)

It is associated with sweat (*Sweda*) glands in *romakūpa*.

कफज खालित्य (*Kaphaja Khālitya*)

In this case the Scalp becomes thick.

पालित्य (Pālitya)

शोकश्रमक्रोध कृतः शरीरोस्म शिरोगतः ।
केशान्सन्दोषः पचति पत्रितं सम्भवथ्यतः ॥

Graying of scalp hair is called as *Pālitya* and it is usually develops due to ageing.

Hair Disorders

Patients may present with too much hair (hirsutism) or too little hair (alopecia)

Tinea capitis

- Fungal scalp infection is becoming increasingly common in urban areas in the UK, where it is most frequently spread by contact between children.
- Clinical features are variable due to associated inflammation with patchy-hair loss and some scaling.
- Affected individuals should have the area scraped and affected hairs plucked for mycological microscopy and culture.

Endothrix (within the hair shaft) infections

- Endothrix (within the hair shaft) infections, e.g. *Trichophyton tonsurans,* cause relatively uninflamed patchy baldness with breakage of the hairs at the skin surface ('black dot').
- There is no fluorescence under Wood's light.

Ectothrix (outside the hair shaft) infections

- Ectothrix (outside the hair shaft) species of fungi, such as Microsporum *audouinii* (anthropophilic), show minimal inflammation
- Microsporum *canis* (from dogs and cats) infections are more inflamed and can be identified by green fluorescence with Wood's light.

- Kerions are boggy, highly inflamed areas of tinea capitis and are usually caused by zoophilic (from animals, e.g. cattle ringworm) species of fungi (e.g. *Trichophyton verrucosum*).

Alopecia areata

- This non-scarring condition appears as sharply defined non-inflamed bald patches, usually on the scalp.
- During the active stage of hair loss, pathognomonic 'exclamation mark' hairs are seen (broken-off hairs 3-4 mm long, which taper off towards the scalp).
- An uncommon diffuse pattern on the scalp is recognised.
- The condition may affect the eyebrows, eyelashes and beard.
- Pitting and longitudinal wrinkling of the nail may be seen.
- The hair usually regrows spontaneously in small bald patches, but the outlook is less good with larger patches and when the alopecia appears early in life or is associated with atopy.
- There is an association of alopecia areata with autoimmune disorders, atopy and Down's syndrome.

Alopecia totalis

- It describes complete loss of scalp hair

Alopecia universalis

- It is complete loss of all hair.

Classification alopecia

Localised	Diffuse
Non-scarring	
Tinea capitis	Androgenetic alopecia
Alopecia areata	Telogen effluvium
Androgenetic alopecia	Metabolic
Traumatic (trichotillomania, traction, cosmetic)	Hypothyroidism Hyperthyroidism Hypopituitarism
Syphilis	Diabetes mellitus
	HIV disease
	Nutritional deficiency
	Liver disease
	Post-partum
	Alopecia areata
	Syphilis
Scarring	
Idiopathic	Discoid lupus erythematosus
Developmental defects	Radiotherapy
Discoid lupus erythematosus	Folliculitis decalvans
Herpes zoster	Lichen planus pilaris Pseudopelade

Androgenetic alopecia

1. Male-pattern baldness is physiological in men over 20 years old, although rarely it may be extensive and develop at an alarming pace in the late teens.
2. It also occurs in females, most obviously after the menopause.
3. The well-known distribution (bitemporal recession and then crown involvement) is described as 'male-pattern' but hair loss in females is often diffuse.

Investigations

The following tests help to determine the cause of non-scarring alopecia.

1. A full blood count
2. ESR
3. Urea and electrolytes
4. Liver and thyroid function tests,
5. Autoantibody profile
6. Syphilis serology

Specialised tests,

7. The hair pluck test in which up to 50 hairs are removed with epilating forceps to determine the anagen:telogen ratio
8. Mycological assessment is advisable in cases of localised hair loss with scaling.
9. In scarring alopecia, scalp biopsy, with direct immunofluorescence, may help to confirm a diagnosis of lichen planus of the scalp or discoid lupus erythematosus.

Hirsuitism

- It is the growth of the terminal hair in a male pattern in females.
- It is one of the most common endocrine disorders affecting less percentage of women.
- The hairiness implies the presence of abnormal androgen action, which may represent a serious or, more likely, a nonserious medical problem.
- Regardless of the etiology, hirsutism can produce mental trauma and emotional anguish.

Causes

1. Endocrine-related causes
   - adrenocortical disorders and ovarian disorders
   - Tumors, Cushing syndrome, and CAH are the adrenocortical causes.
   - Tumors (malignant or benign) and PCOS are the ovarian causes of hirsutism.
2. Idiopathic
3. Medication-related
4. Miscellaneous.

MCQs

1.*Nakhaprabha* is the feature of
   a. *Vataja Khalitya*
   b. *Pittaja Khalitya*
   c. *Kaphaja Khalitya*
   d. *Sannipatika Khalitya*

2. *Agnidagdhabha* is the feature of
   a. *Vataja Khalitya*
   b. *Pittaja Khalitya*
   c. *Kaphaja Khalitya*
   d. *Sannipatika Khalitya*

3. *Swinna siravrita* is the feature of
   a. *Vataja Khalitya*
   b. *Pittaja Khalitya*
   c. *Kaphaja Khalitya*
   d. *Sannipatika Khalitya*

4. दग्धाग्निनेवनिर्लोमासदाहा is the feature of
   a. *Vataja Khalitya*
   b. *Pittaja Khalitya*
   c. *Kaphaja Khalitya*
   d. *Sannipatika Khalitya*

5. जलप्रभम् is seen in
   a. *Pittaja Khalitya*
   b. *Vataja Palitya*
   c. *Vataja Khalitya*
   d. *Kaphaja Khalitya*

Answer

1.	d	2.	a	3.	b
4.	a	5.	b		

●

CHAPTER
59

Shleepada

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

Introduction

Filariasis. *Ślīpada* is a progressive swelling of lower limb which generally develops after the attack of fever. In this indisposition due to solid oedema of the foot and leg the patient becomes hard like stone. Sometimes the indisposition also affects hand and scrotum Elephantiasis-Morbid enlargement of the leg, swelled leg.

श्लीपद हेतु (Ślīpada Hetu)

पारियात्रभवा याश्च विन्ध्यसह्यभवाश्च याः।
शिरोहृद्रोगकुष्ठानां ता हेतुः श्लीपदस्य च॥
C.S.Su. 27/212

Rivers which originates from *Pariyātra* (Western *vindhya* range), *Vindhya, Sahya* ranges causes the diseases of head, heart, *Kuṣṭha,* & *Ślīpada.*

महेन्द्रप्रभवाः श्लीपदोदराणि, हिमवत्प्रभवा हृद्रोगश्वयथुशिरोरोगश्लीपदगलगण्डान्। *S.S.Su. 45/21*

Water of rivers arising from *Mahendra* mountains (North range of Western ghat) produces *Ślīpada & Udara.* Water of rivers arising from *Himvat (Himālaya)* mountains causes *Hṛdroga, Śvayathu, Śiroroga, Ślīpada* and *Galagaṇḍa.*

पुराणोदकभूयिष्ठाः सर्वर्तुषु च शीतलाः।
ये देशास्तेषु जायन्ते श्लीपदानि विशेषतः॥
S.S.Ni. 12/17

Ślīpada develops in those places which have a collection of stagant water and cold climate in all the seasons.

श्लीपद संप्राप्ति (Samprāpti of Ślīpada)

षष्ठी रोहिणी नाम व्रीहिप्रमाणा, ग्रन्थ्यपच्यर्बुदश्लीपदगलगण्डाधिष्ठाना। *S.S.Śā. 4/4*

Sixth layer of skin named *Rohiṇī* equal to one grain of paddy in thickness, is the seat of *Granthi, Apaci, Arbuda, Ślīpada* & *Galagaṇḍa.*

जङ्घासु पिण्डीप्रपदोपरिष्टात् स्याच्छ्लीपदं मांसकफास्रदोषात्।
C.S.Ci. 12/98

Ślīpada manifest by vitiating *Māmsa, Kapha* & *Rakta.* It is characterised by swelling in the calf region, lower extremities & swelling originates from the portion of the leg.

कुपितास्तु दोषा वातपित्तश्लेष्माणोऽधः प्रपन्ना वङ्क्षणोरुजानुजङ्घास्ववतिष्ठमानाः कालान्तरेण पादमाश्रित्य शनैः शोफं जनयन्ति, तं श्लीपदमित्याचक्षते। तत् त्रिविधं–वातपित्तकफनिमित्तमिति॥
S.S.Ni. 12/10

प्रस्थिता वङ्क्षणोर्वादिमधः कायं कफोल्बणाः॥
दोषा मांसास्रगाः पादौ कालेनाश्रित्य कुर्वते।
शनैः शनैर्घनं शोफं श्लीपदं तत्प्रचक्षते॥
A.H.Utt. 29/18-19

Excessively exacerbated *Vāta, Pitta,* & *Kapha* move downwards and get localised in the groins, thighs, foreleg, & calves, gradually reach the foot in due course of time & gives rise to swelling is known as *Ślīpada.*

Nidāna Sevana (Consumption of Etiological factors)
↓
Aggravation of *Kapha pradhan Tridoṣa*
↓
Vitiates *Māmsa* & *Rakta*
↓
Move downwards
↓
Get localised in the groins, thighs, foreleg, calves and gradually reach the foot
↓
Swelling in the lower limbs
↓
Ślīpada

भेद (Bheda)

Ślīpāda is classified into three types namely *Vātaja, Pittaja,* and *Kaphaja.*

त्रीण्यप्येतानि जानीयाच्छलीपदानि कफोच्छ्रयात्।
गुरुत्वं च महत्त्वं च यस्मान्नास्ति विना कफात्॥
S.S.Ni. 12/16

All varieties of *Ślīpada* are produced by *Kapha* because without *Kapha* heaviness & big size never manifest.

श्लीपद लक्षण (Ślīpada Lakṣaṇa)

यः सज्वरो वङ्क्षणजो भृशार्तिः शोथो नृणां पादगतः क्रमेण।
तच्छ्लीपदं स्यात् करकर्णनेत्रशिश्नौष्ठनासास्वपि केचिदाहुः॥
M.Ni. 39/1

Disease *Ślīpada* is characterised by fever; swelling starts from groins and gradually extend to feet associated with pain. Some say it may manifest in other places like hands, ears, eyes, penis, lips and nose.

वातज श्लीपद (Vātaja Ślīpada)

तत्र, वातजं खरं कृष्णं परुषमनिमित्तानिलरुजं परिस्फुटति च बहुशः। *S.S.Ni 12/14*

वातजं कृष्णारूक्षं च स्फुटितं तीव्रवेदनम्।
अनिमित्तरुजं तस्य बहुशो ज्वर एव च॥
M.Ni 39/2

***Vātaja Ślīpada* Signs & Symptoms are as follows :** Affected area becomes rough, blackish, coarse, has various kinds of *Vātaja* pain like piercing, pricking etc, severe pain appearing without any apparent cause, excessive crackness & fever.

पित्तज श्लीपद (Pittaja Ślīpada)

पित्तजं तु पीताव-भासमीषन्मृदुज्वरदाहप्रायं च।
S.S.Ni 12/14

***Pittaja Ślīpada* Signs & Symptoms are as follows :** Affected area becomes yellowish, slightly soft, associated with fever & burning sensation.

कफज श्लीपद (Kaphajā Ślīpada)

श्लेष्मजं तु श्वेतं स्निग्धावभासं मन्दवेदनं भारिकं महाग्रन्थिकं कण्टकैरुपचितं च॥ *S.S.Ni 12/14*

Signs & Symptoms of *Kaphaja Ślīpada* are as follows : Affected area becomes whitish, appears unctuous, has mild pain, heaviness, bigger nodules and studded with throny projections.

साध्यासाध्यता (Sādhyāsādhyatā)

Prognosis of Ślīpada

तत्र संवत्सरातीतमतिमहद्वल्मीकजातं प्रसृतमिति वर्जनीयानि॥
S.S.Ni 12/12

If *Ślīpada* persist for more than one year, growing bigger & bigger resembling like ant hill & exuding fluids should be rejected.

यच्छ्लेष्मलाहारविहारजातं पुंसः प्रकृत्याऽपि कफात्मकस्य।
सास्रावमत्युन्नतसर्वलिङ्गं सकण्डुरं श्लेष्मयुतं विवर्ज्यम्॥
M.Ni. 39/7

Ślīpada manifest due to consumption of foods & habits which aggravates *Kapha, Kaphaja Prakṛti,* exuding bigger in size, severe itching associated with all the symptoms should be rejected for treatment.

उपशय/पथ्य (Upaśaya/Pathya)

Process of Treatment: *Laṅgana*, external application, *Svedana*, *Virecana*, blood-letting and such other therapies as are hot and alleviator of *Kapha* should be used for the treatment of *Ślīpada* (filariasis). (B.R. 45/1)

The following measures and edibles are considered to be beneficial for the patient suffering from the disorder of *Ślīpada*: *Vamana*, *Laṅgaṇa* (fasting), *Rakta Mokṣana* (blood letting), *Svedana*, *Virecana*, *Lepa*, old *Ṣaṣṭhi* variety of rice, *Yava* (barley), *Kulattha*, garlic, *Paṭola*, brinjal, fruits of *Śobhāñjana*, *Kāravellaka*, *Punarnavā*, radish, *Pūtikā* leaves, oil of *Eraṇḍa*, cow urine, *Kaṭu* and the like foods which promote digestive power. (B.R. 45/57-58)

अनुपशय/अपथ्य (Anupaśaya/Apathya)

The following measures and edibles are considered to be harmful for the *Ślīpada* patients: *Piṣṭānna*, *Dugdhavikṛti* (milk products), *Guḍa* (jaggery), meat of animals and birds living in marshy land, sweet juices, river water evolving from the mountains of Mahendra, Sahya and Vindhya, oily products, heavy to digest and *Abhiṣyandi* products. (B.R. 45/59-60)

Filariasis/ lymphatic filariasis

Introduction

1. Filarial worms are tissue-dwelling nematodes.
2. The larval stages are inoculated by biting mosquitoes or flies, each specific to a particular filarial species.
3. The larvae develop into adult worms (2-50 cm long) which, after mating, produce millions or microfilariae (170-320 mm long) that migrate in blood or skin.
4. The life cycle is completed when the vector takes up microfilariae while feeding on humans.
5. In the insect, ingested microfilariae develop into infective larvae for inoculation in humans, normally the only host.
6. Disease is due to the host's immune response to the worms (both adult and microfilariae), particularly dying worms, and its pattern and severity vary with the site and stage of each species
7. The worms are long-lived; microfilariae survive 2-3 years and adult worms 10-15 years.
8. The infections are chronic and worst in individuals constantly exposed to reinfection.

Causes

Infection with the filarial worms Wuchereria bancrofti and Brugia malayi is associated with clinical outcomes ranging from subclinical infection to hydrocele and elephantiasis

Mode of transmission

1. W. bancrofti is transmitted by night-biting culex or anopheline mosquitoes in most areas.
2. The adult worm, 4-10 cm in length, live in the lymphatics, and the females produce microfilariae which circulate in large numbers in the peripheral blood, usually at night.
3. The infection is widespread in tropical Africa, the North African coast, coastal areas of Asia, Indonesia and Northern Australia, the South Pacific islands, the West Indies and also in North and South America.

Pathogenicity of filarial infections depending on site and stage of worms

Worm species	Adult worm	Microfilariae
Wuchereria bancrofti and	Lymphatic vessels-	Blood+ Pulmonary
Brugia malayi *Loa loa*	Subcutaneous+	capillaries++ Blood+
Onchocerca volvulus	Subcutaneous+	Skin+++ Eye+++
Mansonella perstans	Retroperitoneal-	Blood+
Mansonella streptocerca	Skin+	Skin++

(+++ severe; ++ moderate; + mild; - rarely pathogenic)

Pathology

Toxins, released by the adult worm cause lymphangiectasia

↓

The dilatation of the lymphatic vessels leads to lymphatic dysfunction

↓

Leading to lymphatic filariasis, lymphoedema and hydrocele

↓

Death of the adult worm results in acute filarial lymphangitis.

↓

The filariae are symbiotically infected with rickettsia-like bacteria (*Wolbachia* spp.)

↓

Release of lipopolysaccharide from these bacteria contributes to inflammation.

↓

Lymphatic obstruction persists after death of the adult worm.

↓

Secondary bacterial infections cause tissue destruction.

↓

The host response to microfilariae is central to the pathogenesis of tropical pulmonary eosinophilia.

Clinical features

1. Acute filarial lymphangitis presents with fever, pain, tenderness and erythema along the course of inflamed lymphatic vessels.
2. Inflammation of the spermatic cord, epididymis and testis is common.
3. The whole episode lasts a few days but may recur several times a year.
4. Temporary oedema becomes more persistent and regional lymph nodes enlarge.
5. Progressive enlargement, coarsening, corrugation, fissuring and bacterial infection of the skin and subcutaneous tissue develop gradually, causing irreversible 'elephantiasis'.
6. The scrotum may reach an enormous size.
7. Chyluria and chylous effusions are milky and opalescent; on standing, fat globules rise to the top.
8. The acute lymphatic manifestations of filariasis must be differentiated from thrombophlebitis and infection.
9. The oedema and lymphatic obstructive changes must be distinguished from congestive cardiac failure, malignancy, trauma and idiopathic abnormalities of the lymphatic system.
10. *Non-filarial elephantiasis* : Silicates absorbed from volcanic soil can also cause non-filarial elepha-ntiasis.

Diagnosis

In the earliest stages of lymphangitis the diagnosis is made on clinical grounds, supported by eosinophilia and sometimes by positive filarial serology.

Investigations

1. *Full Blood Count (FBC)* :Filarial infections cause the highest eosinophil counts of all helminthic infections.
2. *Blood for Microfilariae :* Microfilariae can be found in the peripheral blood at night
3. Microfilariae are seen moving in a wet blood film or are detected by microfiltration of a sample of lysed blood.
4. *Hydrocele fluid examination* : They are usually present in hydrocele fluid, which may occasionally yield an adult filaria.
5. By the time elephantiasis develops, microfilariae become difficult to find.
6. *Radiography* : Calcified filariae may sometimes be demonstrable by radiography.
7. *Scrotal ultrasound :* Movement of adult worms can be seen on scrotal ultrasound.
8. *PCR-based tests:* PCR-based tests for detection of W. *bancrofti* and B. *malayi* DNA from blood have been developed.
9. *Indirect fluorescence and ELISA* : Indirect fluorescence and ELISA detect antibodies in over 95% of active cases and 70% of established elephantiasis.
10. *Immunochromatographic card tests :* Highly sensitive and specific immunochromatographic card tests for detection of filarial infection are now commercially available; finger prick blood taken at any time of the day can be used for these.

Tropical pulmonary eosinophilia

1. Tropical pulmonary eosinophilia is a complication of Filarial infections seen mainly in India
2. It is due to microfilaria trapped in the pulmonary capillaries and destroyed by allergic inflammation.

Clinical features

1. It is characterised by paroxysmal cough, wheeze and fever.
2. If untreated, this progresses to debilitating chronic interstitial lung disease.

Investigations

1. In tropical pulmonary eosinophilia, serology is strongly positive and IgE levels are massively elevated but circulating microfilariae are not found.
2. The chest X-ray shows military changes or mottled opacities.
3. Pulmonary function tests show a restrictive picutre

MCQs

1. Which layer of skin is the *Adhishtana* of *Shleepada*
   - a. *Tamra* b. *Vedini*
   - c. *Rohini* d. *Mamsadhara*
2. *Dushyas involved in Shleepada*
   - a. *Rakta Mamsa*
   - b. *Rasa Rakta Mamsa*
   - c. *Rakta Mamsa Medas*
   - d. *Mamsa Medas*
3. Types of *Shleepada*
   - a. 3 b. 4
   - c. 5 d. 7
4. *Kandakairupachitam* is the feature of
   - a. *Vataja Sleepada* b. *Pittaja Sleepada*
   - c. *Kaphaja Sleepada* d. *Both a & b*

Answer

1.	c	2.	a	3.	a
4.	c				

CHAPTER

60

Tuberculosis

Prof. Parameswarappa S. Byadgi and Dr. Arunima P.S.

Mycobacterium tuberculosis (MTB) is the causative bacteria for the development of Tuberculosis (TB) and it is part of a complex of organisms including *M. boivs* (reservoir cattle) and M. africanum (Reservoir human).

Epidemiology

There were an estimated 9.2 million new cases reported worldwide and it is estimated that 14.4 million prevalent cases, around one-third of the world's population has latent TB and 1.5 million deaths attributable to TB. The majority of cases occur in the world's poorest nations, who struggle to cover the costs associated with management and control programmes. The resurgence of TB has been largely driven by HIV disease and other immunocompromised diseases.

Etiogy

1. M. Bovis infection arises from drinking non-sterilised milk from infected cows.
2. M. tuberculosis is spread by the inhalation of aerosolised droplet nuclei from other infected patients.

Pathology and pathogenesis

Inhalation of aerosolised droplet nuclei from other infected patients

↓

The organisms lodge in the alveoli

↓

Initiate the recruitment of macrophages and lymphocytes

↓

Macrophages undergo transformation into epithelioid and Langhans cells

↓

Which aggregate with the lymphocytes to form the classical tuberculous granuloma

↓

Numerous granulomas aggregate to form a primary lesion or 'Ghon focus' (a pale yellow, caseous nodule, usually a few mm to 1-2 cm in diameter)

↓

It is situated in the periphery of the lung

↓

Tuberculosis (TB)

Mode of Tuberculosis (TB) spread

Spread to the hilar lymph nodes

↓

Develops similar pathological reaction

↓

The combination of a primary lesion and regional lymph nodes

↓

Primary complex of Ranke

↓

Reparative processes encase the primary complex in a fibrous capsule

↓

This lesion eventually calcifies (limit the spread of bacilli-so-called latent T.B)

↓

It is clearly seen on a chest X-ray

Latent T.B

Lymphatic or haematogenous spread may occur before immunity is established

↓

Seeding secondary foci in other organs

↓

(Lymph nodes, serous membranes, meninges, bones, liver, kidneys and lungs)

↓

The estimated lifetime risk of developing disease after primary infections is 10%

↓

Which may lie dormant for years

Clinical features: Pulmonary disease

1. Chronic cough
2. Haemoptysis
3. Fever
4. Night sweats
5. Malaise
6. Loss of appetite
7. Loss of weight
8. Unresolved pneumonia

9. Asymptomatic(diagnosis on chest x ray)
10. General debility
11. Exudative pleural effusion

Military TB

1. Blood-borne dissemination gives rise to miliary TB, which may present acutely.
2. Characterised by 2-3 weeks of fever, night sweats, anorexia, weight loss and a dry cough.
3. Hepatosplenomegaly may develop and the presence of a headache may indicate coexistent tuberculous meningitis.
4. Auscultation of the chest is frequently normal, although with more advanced disease widespread crackles are evident.
5. Fundoscopy may show choroidal tubercles.
6. The classical appearances on chest X-ray are of fine 1-2 mm lesions (millet seed) distributed throughout the lung fields, although occasionally the appearances are coarser.
7. Anaemia and leucopenia reflect bone marrow involvement.

Clinical features : Extra pulmonary disease

Extra pulmonary tuberculosis accounts for about 20% of cases in those who are HIV-negative but is more prevalent in HIV-positive individuals.

Lymphadenitis

1. Lymph nodes are the most common extra pulmonary site of disease.
2. Cervical and mediastinal glands are affected most frequently, followed by axillary and inguinal; more than one region may be involved.
3. Disease may represent primary infection, spread from contiguous sites or reactivation.
4. Supraclavicular lymphadenopathy is often the result of spread from mediastinal disease.
5. The nodes are usually painless and initially mobile but become matted together with time.
6. When caseation and liquefaction occur, the swelling becomes fluctuant and may discharge through the skin with the formation of a 'collar-stud' abscess and sinus formation.
7. Approximately half of cases fail to show any constitutional features such as fever or night sweats.
8. The tuberculin test is usually strongly positive.
9. During or after treatment paradoxical enlargement, development of new nodes and suppuration may all occur but without evidence of continued infection, rarely, surgical excision is necessary.

Gastrointestinal disease

1. Upper gastrointestinal tract involvement is rare
2. Ileocaecal disease accounts for approximately half of abdominal TB cases.
3. Fever, night sweats, anorexia and weight loss are usually prominent and a right iliac fossa mass may be palpable.
4. Up to 30% of cases present with an acute abdomen.
5. Ultrasound or CT may reveal thickened bowel wall, abdominal lymphadenopathy, mesenteric thickenings or ascites.

Pericardial disease

1. Disease occurs in two forms: Pericardial effusion and constrictive pericarditis.
2. Fever and night sweats are rarely prominent
3. The presentation is usually insidious with breathlessness and abdominal swelling.
4. Pulsus paradoxius, a raised JVP, hepatomegaly, prominent ascites and peripheral oedema are common to both types.
5. Pericardial effusion is associated with increased pericardial dullness and a globular enlarged heart on chest X-ray
6. Constriction is associated with a raised JVP, an early third heart sound and, occasionally, atrial fibrillation; pericardial calcification occurs in around 25% of cases.

Central nervous systems disease

1. Meningeal disease represents the most important form of central nervous system TB.
2. Unrecognized and untreated, it is rapidly fatal.

Bone Tuberculosis

1. The spine is the most common sites for bony TB (Pott's disease)
2. Which usually presents with chronic back pain and typically involves the lower thoracic and lumbar spine.

3. The infection starts as a discitis and then spreads along the spinal ligaments to involve the adjacent anterior vertebral bodies, causing angulation of the vertebrae with subsequent kyphosis.
4. Paravertebral and psoas abscess formation is common and the disease may present with a large (cold) abscess in the inguinal region.

Joint Tuberculosis

1. TB can affect any joint, but most frequently involves the hip or knee.
2. Presentation is usually insidious with pain and swelling
3. Fever and night sweats are uncommon.

Genitourinary disease

Renal tract Tuberculosis

1. Fever and night sweats are rare
2. Mildly symptomatic for many years Hematuria, frequency and dysuria are often present
3. Sterile pyuria found on urine microscopy and culture.
4. In women, infertility from endometritis, or pelvic pain and swelling from salpingitis or a tubo-ovarian abscess occur occasionally.
5. In men, genitourinary TB may present as epididymitis or prostatitis.

Physical examination

1. Lymphadenopathy in TB occurs as painless swelling of 1 or more lymph nodes.
2. Lymphadenopathy is usually bilateral and typically involves the anterior and posterior cervical chain or supraclavicular nodes.
3. Patients with pulmonary TB have abnormal breath sounds, especially over the upper lobes or involved areas.
4. Crackles/ Crepitations/Rales or bronchial breath signs may be noted, indicating lung consolidation.

Signs of extrapulmonary TB differ according to the tissues involved.

- Confusion
- Coma
- Neurologic deficit
- Chorioretinitis
- Lymphadenopathy
- Cutaneous lesions

Diagnosis

1. The presence of an otherwise unexplained cough for more than 2-3 weeks, particularly in an area where TB is highly prevalent, or typical chest X-ray changes should prompt further investigation.
2. Direct microscopy of sputum (induced with nebulised hypertonic saline if not expectorating) at least 2 but preferably 3 (early morning samples). The probability of detecting acid-fast bacilli is proportional to the bacillary burden in the sputum (typically positive when 5000-10,000 organisms are present). Tuberculous bacilli are difficult to stain due to their substantial lipid-rich wall

   The most effective techniques are the Ziehl-Neelsen and rhodamine-auramine stains

   Rhodamine-auramine stains causes the tuberculosis bacilli to fluoresce against a dark background and is easier to use when numerous specimens need to be examined.
3. A positive smear is sufficient for the presumptive diagnosis of TB but definitive diagnosis requires culture.

   Smear-negative sputum should also be cultured, as only 10-100 viable organisms are required for sputum to be culture-positive.

Blood investigations

Raised ESR, CRP, Anaemia etc

Tuberculin skin test (low sensitivity/ specificity; useful in primary or deep seated infection)

Extrapulmonary

Fluid examination -CSF, ascitic, pleural, pericardial, synovial fluid

Tissue biopsy.

MCQs

1. Which of these is the culture medium for Mycobacterium tuberculosis?
   a. Wilson blair medium
   b. Lowenstein–Jensen medium
   c. Mac Conkey's medium
   d. Dubo's culture medium
2. World Health Organization recommended a control strategy for TB known as:
   a. DOTS
   b. Gene therapy
   c. Morphine
   d. MCT
3. The first person who discovered Mycobacterium tuberculosis was
   a. Louis Pasteur
   b. Robert Koch
   c. Edward Jenner
   d. Hans Christian Gram

Answer

1.	b	2.	a	3.	b